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THE JOURNAL OF THE MEDICAL SOCIETY OF NEW JERSEY JANUARY 1986

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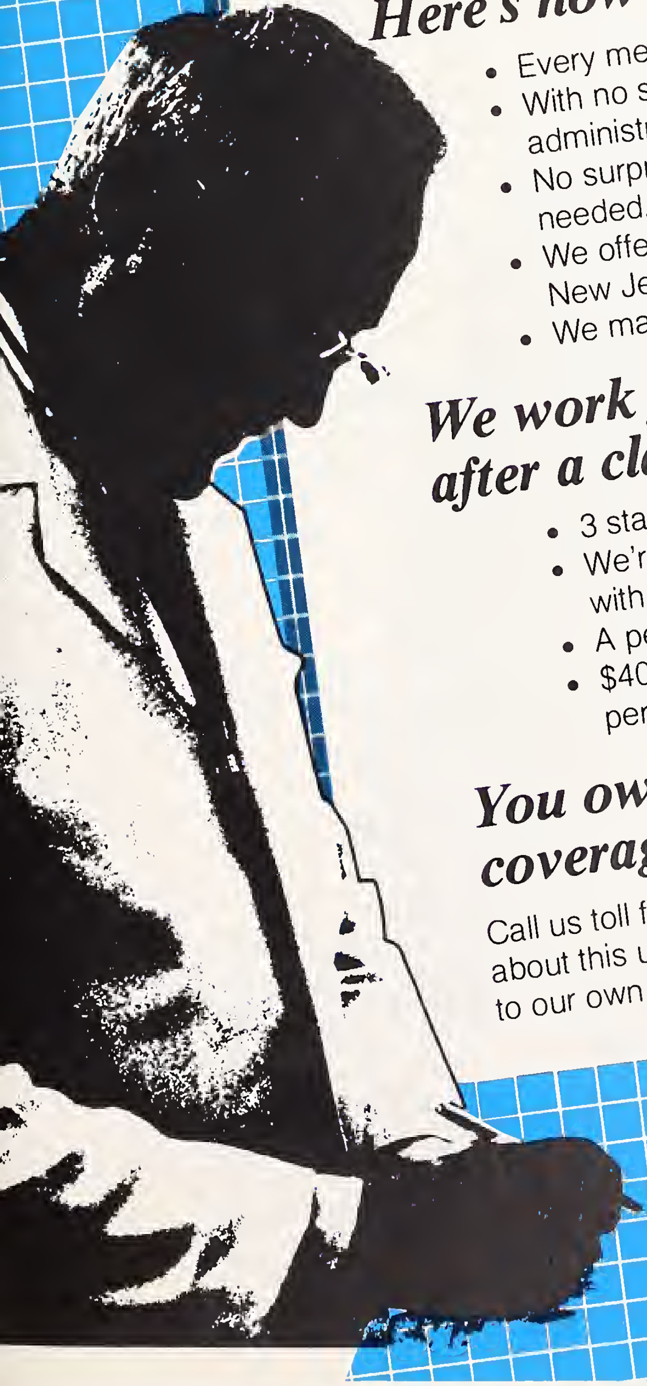

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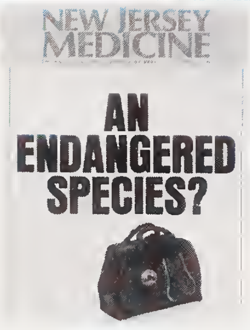

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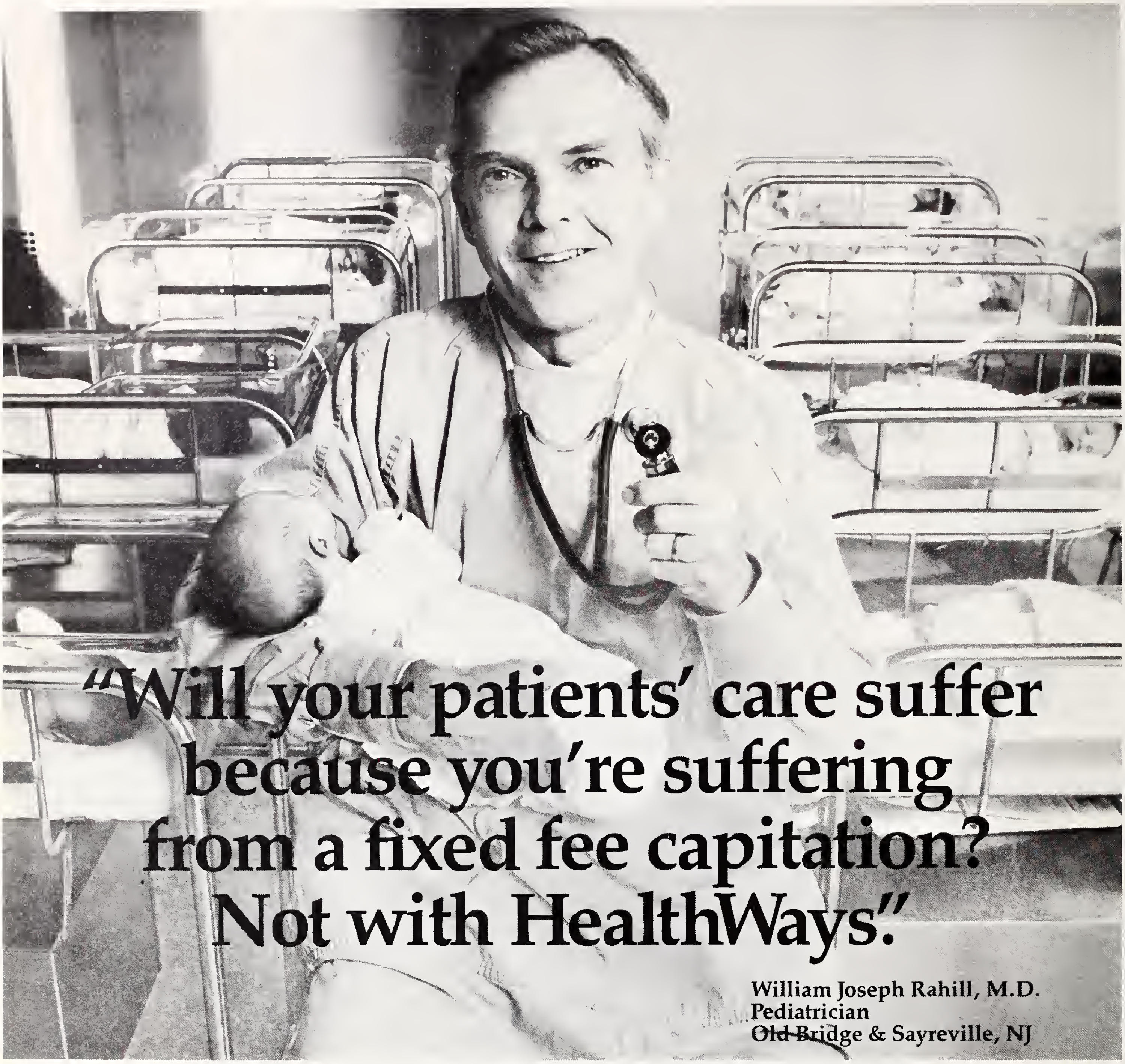
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MEMBERSHIP NEWSLETTER

MSNJ

THE MEDICAL SOCIETY OF NEW JERSEY VOLUME 29

HOME HEALTH SERVICE ABUSE

The AMA House of Delegates adopted Resolution 35 at its 1985 Annual Meeting; the Resolution reads as follows:

Resolved, that the American Medical Association call upon the nation's physicians to write or carefully review all initial and renewal orders for home health services and approve only those that are medically indicated; and be it further

Resolved, that the AMA urge physicians to report to appropriate payment agencies and regulatory authorities cases of abusive practices by home health agencies; and be it further

Resolved, that the AMA urge physicians not to authorize the provision of home health services to any patient with whom he or she is not professionally involved in providing care.

Adoption of the Resolution was motivated by concern for assuring that the services provided to any patient by a home health agency are only those adjudged appropriate to the patient's needs by a physician involved in that patient's care.

NEW SECRETARY FOR ACURP

Thomas J. Connolly, Jr., M.D., a general practitioner from Jersey City, was elected Secretary of the American College of Utilization Review Physicians. For the past 33 years, Dr. Connolly has been affiliated with St. Francis Hospital, Jersey City, where he is a past chairman of the Quality Assurance Committee. A past president of the Hudson County Medical Society and a member of its Executive Committee for the last ten years, Dr. Connolly is treasurer of the Metropolitan Peer Review Organization and secretary of JEMPAC. Dr. Connolly is certified by the American Board of Quality Assurance and Utilization Review Physicians.

The College, a national nonprofit association, educates physicians, other health-care professonals, and the general public in cost containment, risk management, prospective payment systems, utilization review (processes used to evaluate the allocation of health-care resources), and related topics.

ELDER ABUSE

There is a shocking crime on the rise that you may not be aware of: elder abuse. Grown sons or daughters actually are beating up their elderly parents. And it is happening more and more.

Abuse of the elderly is harder to pin down than child abuse. Only about one in five cases is ever reported. That's because many abused adults are embarrassed or ashamed. Some even think they deserve to be punished.

The typical abuser is a man, between 40 to 60, who is part of the victim's immediate family. The average victim is a woman, over the age of 75, who is in poor health and can't move around very well. Abuse can be physical, mental, or financial. But physical abuse is most common when victim and abuser live in the same house.

Union County is cracking down on elder abuse. Prosecutor John Stamler has stopped accepting plea bargains in cases of violent crime against senior citizens. Now he's setting up a hotline for reporting abuse, and assigning a staff member to follow up.

But Stamler thinks police need to be made more aware of elder abuse. Sensitivity training helped police cope with child abuse, domestic violence, and rape victims. Stamler thinks it will also help with elder abuse.

Governor Kean also is looking into the problem. He has appointed a task force to investigate and make recommendations. That is a step in the right direction but the report won't be ready until next year. We believe action is needed now. We urge other communities to follow Union County's lead and crack down on elder abusers immediately. The victims need our support. Stopping elder abuse deserves our highest priority.

FAIRNESS DOCTRINE

The Fairness Doctrine is the listener's bill of rights. Established in 1949, the Fairness Doctrine requires broadcasters to devote "a reasonable percentage of their time to covering important issues in the community and the nation." And it requires that television and radio stations allow "for the expression of the contrasting views of all responsible elements in the community on the various issues which arise." The Fairness Doctrine is based on two sound principles: 1) Radio and television stations are trustees of the public; 2) The public interest is served by free and open public debate, with citizens hearing as many points of view as possible.

Now, the Federal Communications Commission (FCC) wants to scrap the Fairness Doctrine contending it "chills and coerces speech and inhibits coverage of major issues." Secondly, the FCC argues that new communications technologies eliminate the need for government action to ensure balanced news coverage.

PROPOSED RULE: TIMELY CLAIMS

The Division of Medical Assistance has submitted a proposed rule which will appear in a future issue of the *New Jersey Register.* The proposal intends to place a time limit on applications that are submitted to the Division's Retroactive Eligibility Unit. Under the proposed rule, Medicaid patients will be required to submit their applications (for retroactive coverage) within six months from the date of application for assistance (AFDC, SSI).

Applications not submitted within the prescribed time frames will not be processed and will be returned to the applicant. The applicant would have the right to request a hearing if he/she disagreed with the Division's denial.

PROSPECTIVE RULE: VISION CARE MANUAL—BILLING PROCEDURES

The Division of Medical Assistance has proposed to readopt N.J.A.C. 10:62-3, which is the Billing Procedures Subchapter of the Vision Care Manual. There are no changes upon readoption, which is being processed to comply with Executive Order No. 66(1978).

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Dear Doctor:

The Medical Society of New Jersey Auxiliary is an active, vital organization whose accomplishments are many and varied. When our accomplishments have fallen short of our goals, we realize that the limitation of manpower often is the reason.

If your spouse is not a member of the Medical Society of New Jersey Auxiliary you both may be unaware of the Auxiliary's efforts on the state level to work for the passage of Medical Society of New Jersey-sponsored liability bills presently in the Senate. Do you know that the Auxiliary is sponsoring and establishing self-help groups for families suffering the stress of malpractice litigation? Are you both aware of the Impaired Spouse Committee which is working to help those among us who are in need?

These are but a few of the projects to which the Medical Society of New Jersey Auxiliary has committed itself. Your spouse can become better informed about these and other issues by becoming a member through your component medical society or by becoming a member-at-large of the State and National Auxiliaries.

I would appreciate your taking the time to discuss Auxiliary participation with your spouse. You both would benefit.

Please complete the form below and send it along with your check in the amount of $20 (payable to "MSNJ Auxiliary") to the Medical Society of New Jersey Auxiliary, Two Princess Road, Lawrenceville, New Jersey 08648. This will provide your spouse with all state and national publications to help be better informed of the issues important to the practice of medicine today. If you are a member of a County Medical Society where there is an active Auxiliary, your spouse will be contacted for County Auxiliary membership.

Thank you for taking the time to read this message. We are all working in the interest of medicine!

Very sincerely yours,

Mrs. Charles J. Moloney (Bernardine)
President

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FINAL 1984-1985 LEGISLATIVE UPDATE

Status of Legislation on Which MSNJ Has Taken an Active Position

Bill	Subject	MSNJ Position	Committee	Present Status
S-70 Codey	To change requirements for eligibility to be Commissioner of Health	Active Opposition	Senate: IHW	In committee
S-111 Bubba	To require a physician to present surgical consent forms at the time surgery is agreed upon	Active Opposition	Senate: LIP	In committee
S-243 Hagedorn	To provide procedures for involuntary civil commitment of the mentally ill	Active Opposition	Senate: IHW	In committee
S-424 Dorsey	To provide for the abolishment of certain state agencies for specific periods of time and to create a Joint Legislative Committee on Regulatory Agencies	Active Support	Senate: SGF&IR&VA	In committee
S-607 Bassano	To require the padding of all metal bars in school buses with energy-absorbing materials to be included in the State Board of Education's rules and regulations	Active Support	Senate: LPS&D	In committee
S-723 Brown	To require the State Department of Health to prepare a booklet on breast cancer and require physicians to give a copy of the booklet to breast cancer patients	Active Opposition	Senate: IHW	In committee
S-752 Hirkala	To provide that certain information of hospitals and long-term health care facility review committees be considered confidential	Active Support	Senate: IHW	In committee
S-754 Hirkala	To provide immunity from civil liability to members of hospital governing bodies in connection with certain review actions	Active Support	Senate: J	In committee
S-1067 Feldman	To establish and license two categories of social workers	Active Opposition	Senate: IHW	In committee
S-1079 Dumont	Provides for a 3-year statute of limitations, except for fraud, intentional concealment, or nontherapeutic or diagnostic purpose. Minors have until age 11 for any injury prior to age 8	Active Support	Senate: J	In committee
S-1112 Hurley	To establish a cap on the amount of damages which may be awarded for pain and suffering in certain civil actions	Active Support	Senate: J	In committee
S-1135 Dalton	To eliminate certain sources of windfall benefits in personal injury and wrongful death actions	Active Support	Senate: J	In committee

LEGISLATIVE UPDATE

Status of Legislation on Which MSNJ Has Taken an Active Position

Bill	Subject	MSNJ Position	Committee	Present Status
S-1140 Feldman	To impose certain requirements on the plaintiff in a medical malpractice proceeding	Active Support	Senate: J	In committee
S-1182 Saxton	To establish Auto Insurance Medical Fee Schedule Board	Active Opposition	Senate: LIP	In committee
S-1190 Feldman	To authorize a court to award reasonable attorney's fees to a party who prevails against a frivolous claim for relief in a civil action	Active Support	Senate: J	In committee
S-1775 Orechio	Allows chiropractors who are licensed to practice by written examination in any state and who have completed 4,300 classroom hours in a chiropractic school to become licensed to practice by virtue of a clinical examination	Active Opposition	Senate: LIP	Signed into law as c. 255 P.L. 1985
S-1840 Cardinale	Exempts the private practice of medicine from the certificate of need law	Active Support	Senate: IHW	In committee
S-1846 Garibaldi	Provides that cancers caused by exposure to heat, cold, radiation, or a known carcinogen which manifest themselves in active police or firemen are assumed to be accidental and work-related disabilities	Active Opposition	Senate: SGF&IR&VA	In committee
S-2019 Lynch	Creates the profession of contact lens dispensing and fitting for nonphysicians and nonoptometrists. Licensure and regulation would be through the Board of Examiners of Ophthalmic Dispensers and Ophthalmic Technicians. A prescription by a licensed optometrist or physician would be required	Active Opposition	Senate: LIP	Passed in Senate; in Assembly Committee CI
S-2116 Bassano	Requires physician to inform women undergoing abortions of availability of fetal anesthesia whenever the fetus to be aborted is viable, i.e. able to live outside the womb with or without artificial means of support. Act is not applicable during an emergency, or if the anesthesia decreases the possibility of survival of fetus, or if the anesthesia to the mother will also abolish organic pain to the aborted fetus	Active Opposition	Senate: LIP	Bill referred to Senate Judicial Committee

LEGISLATIVE UPDATE

Status of Legislation on Which MSNJ Has Taken an Active Position

Bill	Subject	MSNJ Position	Committee	Present Status
S-2159 Hagedorn	Substantially alters the involuntary commitment law. Significantly, certification may be effected by psychologists, nurses, or social workers who have conducted examination within the three days prior to certification	Active Opposition	Senate: J	In committee
S-2379 Costa	Provides that consulting physicians are not to be reimbursed unless the patient has agreed, in writing, prior to consultation. Substantial incapacity of patient and mandatory consultation as a result of law, hospital rule, or insurance contract are excepted	Active Opposition	Senate: IHW	In committee
S-2544 Bubba	Requires written consent to surgery except in cases of physical or mental incapacity or emergencies. The State Board, after consultation with the Health Dept., MSNJ, and NJHS, shall prescribe the consent forms which shall be used by physicians	Active Support	Senate: LIP	In committee
S-2711 Hagedorn	Requires a second physician to be present at any termination of pregnancy procedure if the attending has determined the unborn fetus is 24 weeks of age or older. The second physician is to provide immediate care to the child if it is born alive	Active Opposition	Senate: IHW	In committee
S-2712 Hagedorn	Requires the SBME to conduct a study as to when fetal viability occurs so that the conduct of physicians may be governed accordingly. Having conducted such a study, the Board is to promulgate guidelines regarding the termination of pregnancy	Active Opposition	Senate: IHW	In committee
S-2740 Bassano	Deletes the requirement of a certificate of need for cardiac surgical centers. The hospital still would have to comply with Dept. of Health rules and regulations governing these units	Active Support	Senate: IHW	In committee
S-2869 Codey	Amends certificate of need to include physicians whenever a health service has been regionalized by regulation of the Dept. of Health. Regulation would terminate within three years, at which time, the Commissioner could readopt the regulation	Active Opposition	Senate: IHW	Out of committee; 2nd reading

LEGISLATIVE UPDATE

Status of Legislation on Which MSNJ Has Taken an Active Position

LEGISLATIVE ALERT

S-2869-Codey—Health Care Facilities Act/Certificate of Need. S-2869 has been reported out of committee. This bill presents a clear and present danger to the private practice of medicine. Every member of the Medical Society of New Jersey should write and call their Senator and Assemblymen and request a "NO" vote on this bill.

Bill	Subject	MSNJ Position	Committee	Present Status
S-2922 Hagedorn	Creates a commission to study the adequacy of rates allowed for nursing homes and the quality of care rendered to patients in nursing homes	Active Support	Senate: IHW	In committee
S-3203 O'Connor	Creates a separate Board of Chiropractic Examiners within the Division of Consumer Affairs	Active Opposition	Senate: LIP	In committee
S-3302 Dumont	Exempts public entities and public employees from liability for injuries caused by neglect contamination of the environment	Active Opposition	Senate: NRA	In committee
A-115 Otlowski	To authorize optometrists to prescribe a limited number of eye medications	Active Opposition	Assembly: HERP	In committee
A-708 Hardwick	To create a temporary committee to study Diagnosis Related Group System used for setting hospital rates and appropriate $45,000	Active Support	Assembly: CHHS	In committee
A-926 Weidel	To create an Automobile Insurance Fee Schedule Board in the NJ Dept. of Insurance to adopt a reasonable fee schedule for medical services arising from passenger automobile accidents	Active Opposition	Assembly: BI	In committee
A-960 Miller	To permit individual patients or insurers to appeal hospital bills in cases of assignments under DRG	Active Support	Assembly: CHHS	In committee
A-963 Miller	To eliminate DRG methodology and replace it with one to be devised by the New Jersey Department of Health in one year	Active Support	Assembly: CHHS	In committee
A-1056 Muziani	To require attending physicians and certified nurse midwives to inform pregnant women of any drugs expected to be used during pregnancy and the possible effects thereof	Active Opposition	Assembly: CHHS	Out of Senate committee; 2nd reading

LEGISLATIVE UPDATE

Status of Legislation on Which MSNJ Has Taken an Active Position

Bill	Subject	MSNJ Position	Committee	Present Status
A-1158 Herman	To require licensed physicians or chiropractors to notify the State Board of Medical Examiners if they accept Medicare assignments	Active Opposition	Assembly: HERP	In committee
A-1471 Garvin	To require a physician to obtain, in advance, the patient's authorization to perform a biopsy or mastectomy	Active Opposition	Senate: IHW	Signed into law as c. 1985 P.L. 1984
A-1541 Karcher	To provide for the licensing of persons who practice hypnosis and limit the practice, teaching, or preparation of hypnosis tapes to licensed practitioners	Active Opposition	Assembly: HERP	Out of committee; 2nd reading; amended
A-1559 Garvin	Requires Dept. of Health and the State Board of Medical Examiners to prepare a booklet on all aspects of the treatment of breast cancer. Physicians are required to distribute the book to appropriate patients and to discuss it with them	Active Opposition	Assembly: CHHS	Passed in Assembly. In Senate IHW Committee
A-1682 Muhler	Creates a seven-person Board within the Department of Human Services vested with the authority to release or continue the commitment of persons who were found incapable of standing trial or not guilty by reasons of insanity. A Superior Court judge currently makes these decisions	Active Opposition	Assembly: CHHS	In committee
A-1870 Garvin	Amends existing statutes to require either seat belts or restraint systems for all children under ten in a motor vehicle. Current law applies to children under age five	Active Support	Assembly: CI	In committee
A-1918 Miller	Provides that collateral sources of payment shall be used to offset damages. Further provides structured payments may be used at the request of either party if the future damages exceed $100,000	Active Support	Assembly: JUD	In committee
A-1919 Miller	Provides a $100,000 cap on damages for pain and suffering to personal injury actions	Active Support	Assembly: JUD	In committee
A-1921 Miller	Establishes a 3-year statute of limitations in tort actions against health care providers. Children would have until age 11 to bring action for injuries prior to age 8. After age 8, they come under the 3-year standard. Fraud, concealment, and unintentional foreign bodies "toll" the statute	Active Support	Assembly: JUD	In committee

LEGISLATIVE UPDATE

Status of Legislation on Which MSNJ Has Taken an Active Position

Bill	Subject	MSNJ Position	Committee	Present Status
A-1922 Miller	Provides that within 60 days of filing a medical malpractice complaint, the plaintiff must supply an affidavit, by a qualified expert, that reasonable cause exists to believe malpractice occurred	Active Support	Assembly: JUD	In committee
A-2106 Paterniti	Requires nursing homes with 100 beds or more to dedicate 10 percent to indigent, 10 percent to complex care, and 10 percent for medical day care services	Active Opposition	Assembly: CHHS	In committee
A-2115 Otlowski	Permits optometrists to use topical drugs for treatment and diagnostic purposes	Active Opposition	Assembly: CHHS	Returned to committee
A-2122 Cooper	Establishes seating restraint and emergency exit requirements for school buses	Active Support	Assembly: ED	In committee
A-2140 Rooney	Provides that a judge may order a juvenile offender to be examined by a physician, optometrist, or audiologist	Active Opposition	Assembly: JUD	Out of Senate committee; 2nd reading
A-2293 Kalik	Appropriates $9.45 million for the UMDNJ Osteopathic School in Stratford	Active Opposition	Assembly: RFA	Signed into law as c. 4 P.L. 1985
A-2304 Brown	To require that the driver and passengers in the front seat of an operating passenger automobile wear the safety seat belt provided	Active Support	Assembly: LPSD	Signed into law as c. 179 P.L. 1984
A-2757 Kern	Empowers the SBME to license and regulate nurse-midwives. Creates five-member Nurse-Midwives Examining Committee to license and regulate nurse-midwives. Committee would have stipulated, delegated powers from Board and would function independent of Board	Active Opposition	Assembly: HERP	In committee
A-2889 Herman	Provides for separate licensing board to register physician assistants who would function under the supervision of a licensed physician at the 2:1 ratio	Active Opposition	Assembly: CHHS	In committee
A-3310 Hardwick	Provides for structured payments in cases exceeding $100,000 liability in future damages. Prohibits punitive damages in medical malpractice cases	Active Support	Assembly: JUD	In committee
A-3111 Hardwick	Requires the Court to award attorneys' fees to the prevailing party if the other side has been guilty of frivolous actions	Active Support	Assembly: JUD	In committee

LEGISLATIVE UPDATE

Status of Legislation on Which MSNJ Has Taken an Active Position

Bill	Subject	MSNJ Position	Committee	Present Status
A-3254 Muziani	Directs the SBME to conduct a scientific study to determine when fetal viability occurs	Active Opposition	Assembly: CHHS	In committee
A-3255 Muziani	Requires a second physician to be present when a physician terminates a pregnancy involving a live fetus of 24 weeks or older	Active Opposition	Assembly: CHHS	In committee
A-3397 Bocchini	Allows an emergency medical technician to serve on a mobile unit in place of a paramedic or registered nurse	Active Opposition	Assembly: CHHS	Passed in Assembly; in Senate committee
A-3469 Kern	Provides for local planning councils to replace the health systems agencies. These would be funded by an assessment levied against all NJ hospitals	Active Opposition	Assembly: CHHS	In committee
A-3593 Zangari	Creates a separate Board of Chiropractic Examiners within the Division of Consumer Affairs	Active Opposition	Assembly: HERP	In committee
A-3558 Kosco	Creates a commission to study the certificate of need process. The commission would consist of 15 members; two would be physicians. Their report is to be submitted to the legislature and governor	Active Opposition	Assembly: CHHS	In committee
A-3646 Vainieri	Prohibits physicians from performing elective surgery unless the patient has secured a second surgical opinion prior to the surgery	Active Opposition	Assembly: CHHS	In committee
A-3700 Muziani	Requires physicians to advise pregnant patients of the effects of alcohol and the effects of certain drugs on a fetus. The woman shall sign a form confirming the advice was given. The physician is to retain the original and provide patient with copy	Active Opposition	Assembly: CHHS	In committee
A-3824 Franks	Creates a study commission to review the regulatory process. The commission is to determine whether the regulatory functions of the state agencies are necessary, straightforward, nonduplicative, of minimal expense to the regulated entity, and adequate to protect the public interest	Active Support	Assembly: SG,CS,E&VA	In committee
A-4016 Pelly	Establishes a 15-member Impaired Health Professionals Study Commission; Medical Society of New Jersey is to designate 1 of the members	Active Support	Assembly: CHHS	In committee

LEGISLATIVE UPDATE

Status of Legislation on Which MSNJ Has Taken an Active Position

Bill	Subject	MSNJ Position	Committee	Present Status
A-4119 Doria	Creates a licensing board of chiropractic. Transfers all functions and responsibilities of the SBME regarding chiropractic to the new board. Removes the place for a chiropractor from the medical board	Active Opposition	Assembly: HERP	In committee
AR-31 Herman	Requests the Commissioner of Health to educate the public, particularly young people, about the dangers of cigarette smoking	Active Support	Assembly: CHHS	In committee
AR-146 Shusted	Requests the Assembly to consider the recommendations of the American Academy of Physicians in establishing a federal physician DRG program	Active Opposition	Assembly: CHHS	In committee
AR-147 Shusted	Requests the Assembly to consider the recommendations of the NJ Academy of Family Physicians before implementing a physician DRG program in New Jersey	Active Opposition	Assembly: CHHS	In committee

Senate Committee Key

ED:	Education
IHW:	Institutions, Health and Welfare
J:	Judiciary
LIP:	Labor, Industry and Professions
LPS&D:	Law, Public Safety and Defense
NRA:	Natural Resources and Agriculture
SGF&IR&VA:	State Government, Federal & Interstate Relations & Veterans Affairs

Assembly Committee Key

BI:	Banking and Insurance
CHHS:	Corrections, Health and Human Services
CI:	Commerce and Industry
ED:	Education
HERP:	Higher Education and Regulated Professions
JUD:	Judicial
L:	Labor
LPSD:	Law, Public Safety and Defense
RFA:	Revenue, Finance and Appropriations
SG,CS,E&VA:	State Government, Civil Service, Elections, and Veterans Affairs

LOYAL SUPPORT: Scores of physicians have responded to MSNJ's Medical Action Committee (MedAC) drive to improve medicine's participation in the legislative election campaigns. MedAC will amplify on ongoing state activities through JEMPAC. Major issues facing medicine are malpractice and general tort reform and the application of certification of need to physicians' practices. On behalf of all our members, President Ralph J. Fioretti, M.D., would like to thank the many who have responded generously, and those who we are sure will join them.

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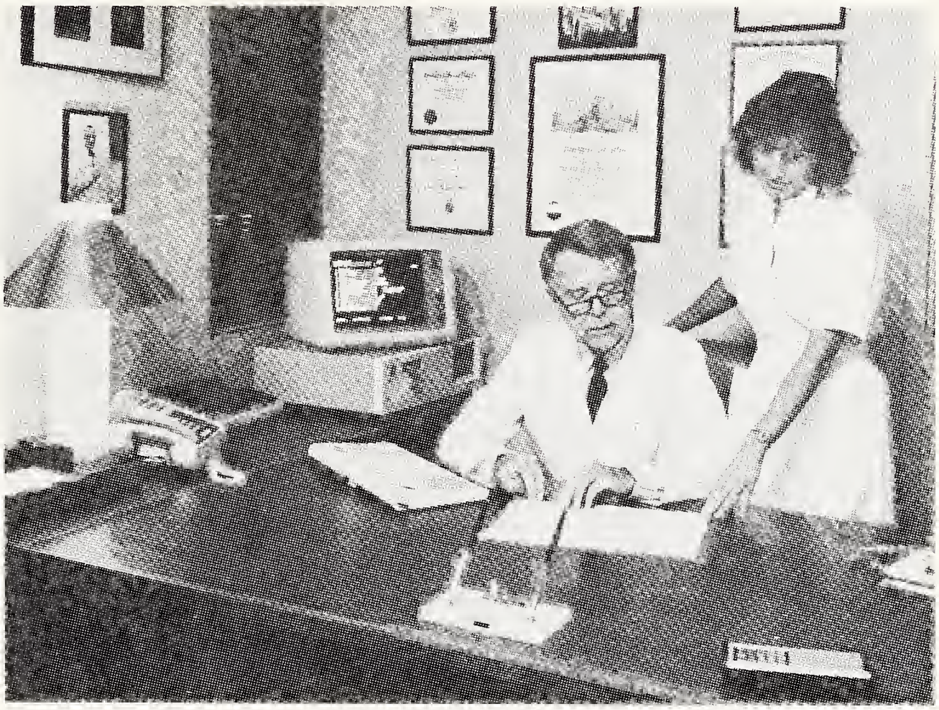

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PROFESSIONAL LIABILITY COMMENTARY*

Patient Followup and Damages

Patient Followup; California Law Limiting Damages; Supreme Court Update

Do your office procedures include a mechanism for followup when a patient misses an appointment? After you refer a patient to another physician, do you ever verify that the patient was indeed seen by the consultant and determine the results of that consultation? Do you, as a consultant, always contact the referring physician to discuss your findings regarding the patient? If you order diagnostic tests on an office patient, do you or your staff followup to confirm that the tests were performed and that the test results were communicated to you, entered into the patient's chart, and, ultimately, related to the patient?

If you answered "no" to any of the above, it is time to establish and implement routine office procedures in this regard.

When a patient misses an appointment, it is a very good practice to have an established mechanism whereby your office staff attempts to contact the patient (preferably by phone) to determine why the patient was unable to keep the appointment and to reschedule it. These attempts to contact the patient should be documented in the record, even if the phone call(s) went unanswered and the patient never was reached.

This followup is important with patients who, in your opinion, require careful, frequent monitoring because of an existing medical condition or present drug therapy. It is arguable that, should subsequent litigation develop, your failure to followup on a missed appointment by such a patient could be characterized as a breach in the standard of care and, therefore, negligence. In these cases, if several phone calls are unsuccessful, a letter to the patient may be advisable. Obviously, a missed appointment for a yearly physical, for example, is a much different situation. However, while not as imperative or as critical to the patient's well-being, followup by your staff in such a nonemergent case only can help strengthen your rapport with the patient, and telephone followup also is recommended in these instances.

A physician has the duty to consult with another physician when he/she knows or reasonably should know that the patient's condition is beyond his/her knowledge, ability, or capacity to treat. The referring physician's duty to the patient does not end with the consultation, and he/she continues to maintain control of the case throughout the consultation process; however, both the referring physician and the consultant have specific followup responsibilities with regard to the patient in question. A referring physician has the responsibility to see that the consultation did indeed occur and to obtain the results of that consultation, inform the patient of those results, and act upon the information derived from the consultation. All aspects of the consultation process—from the physician's rationale for obtaining the consult through the actions taken or not taken based on the consultant's recommendations—should be documented thoroughly in the patient's chart. The consultant also has the responsibility to communicate the results of his/her consultation to the referring physician. Too often, PMSLIC's Claims Committee has reviewed cases where a breakdown in the consultation process led to serious problems for the patient and, ultimately, for the involved physicians when litigation resulted. Followup is especially important when a consultation is deemed necessary because the presence of a neoplastic process is suspected. Any delay in diagnosis or treatment of a neoplasm due to the lack of followup by either the referring or consulting physician can result in litigation based on the increased chance of metastasis and the shortened life expectancy.

When ordering diagnostic tests such as laboratory or radiologic studies on an office patient, it also is important to have a mechanism to assure that the tests were performed. Often, patient noncompliance can be a factor with regard to diagnostic testing. A patient may delay having a test performed for a variety of reasons—anxiety, illness, lack of transportation, funds, or refusal to go for testing. While a patient may be found to be contributorily negligent if a delayed diagnosis or treatment can be at least partially attributed to the patient's own delay in having an ordered test carried out, juries readily accept the argument that the physician better understands the consequences of a patient's noncompliance. Patient delay or noncompliance effectively can be avoided by the physician explaining the necessity for the specific test to the patient at the time it is ordered. If the patient understands the importance of the test findings with respect to his/her present condition, noncompliance will be a less frequent occurrence. A followup procedure should be developed by the physician and his/her office staff to ensure that all diagnostic test results are seen by the physician upon receipt in the office. Malpractice actions often have resulted after a lab or x-ray report was filed in a patient's chart but never was reviewed by the physician through some oversight on the part of the physician or staff. *(Patient Rx Newsletter*, Pennsylvania Medical Society Liability Insurance Company, September 1985, Vol. 7, No. 5).

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*This item from the Department of Professional Liability Control, MSNJ, was prepared by James E. George, M.D., J.D., and A. Ronald Rouse, who are, respectively, Director of the Department and Director of Special Projects.

CALIFORNIA LAW LIMITING DAMAGES IN MALPRACTICE SUITS IS CONSTITUTIONAL

A provision in the California Medical Injury Compensation Reform Act that limits the amount of damages for noneconomic losses to $250,000 was constitutional, the California Supreme Court ruled.

A 34-year-old patient employed by the California State Legislature felt a brief pain in his chest as he was riding his bicycle to work on February 21, 1976. He noticed a similar brief pain the next day while he was jogging, and again three days later while walking after lunch. He made an appointment with his regular physician, who was employed by an HMO. He was examined by a nurse-practitioner, who consulted with her physician consultant. When she returned to the room, she advised him that they believed his pain was due to muscle spasm. The physician prescribed Valium® and the patient went home.

That night he awoke with severe chest pains. His wife took him to the HMO emergency room, where he was examined by a physician. The physician ordered a chest x-ray and, on the basis of his examination and the x-ray results, the physician concluded that the patient was experiencing muscle spasms. He gave him an injection of Demerol® and a prescription for codeine-containing medication.

The patient went home, but continued to experience intermittent chest pain. About noon the same day, the pain became more severe and constant and he returned to the HMO emergency room. Another physician initially believed that his problem was of muscular origin, but he directed that an electrocardiogram (EKG) be performed. The EKG showed that the patient was suffering from an acute myocardial infarction. He then was transferred to the cardiac care unit. He returned to his job part-time about seven months later and full-time in September 1977. At that time he had been permitted to return to virtually all of his prior recreational activities.

In a malpractice action against the HMO, the patient's expert witness testified that the HMO should have performed an EKG during the patient's first two visits to his physician and to the emergency room. He testified that if an EKG had been ordered at those times, it could have revealed the patient's imminent heart attack and treatment could have been administered that might have prevented or minimized the heart attack. He testified that a large portion of the patient's heart muscle had died, reducing his life expectancy by about one-half.

The HMO's expert witnesses indicated that an EKG would not have shown that a heart attack was imminent and that because of the severe disease in the patient's coronary arteries, the heart attack could not have been prevented even if it had been known that it was about to occur.

A jury found in favor of the patient and awarded $24,733 for lost wages, $63,000 for future medical expenses, $700,000 for wages lost in the future as a result of the reduction in his life expectancy, and $500,000 in noneconomic damages for pain, suffering, physician impairment, and other intangible damages.

A trial court reduced the noneconomic damages to $250,000 pursuant to the Medical Injury Compensation Reform Act. The court reduced the award for past lost wages by deducting the amount he had already received in disability payments and ordered the HMO to pay the first $63,000 of any future medical expenses not covered by the patient's medical insurance. The trial court also declined to order the award for future lost wages and economic damages to be paid periodically.

Affirming the decision, the California Supreme Court upheld the constitutionality of the limitation of noneconomic damages in medical malpractice cases to $250,000. The provision was rationally related to the state objective of reducing the costs of malpractice defendants and their insurance companies and did not violate due process. It did not violate equal protection rights on the ground that it discriminated between medical malpractice victims and other tort victims, the court said.

Further, the court said that an additional provision of the Act that modified the collateral source rule was not unconstitutional. Under the collateral source rule, payments by third parties such as medical insurance or disability benefits could not be considered by a jury in calculating a patient's damages. Under the Act, payments received from collateral sources could be considered in calculating damages. (Fein *v* Permanente Medical Group, 695 P.2d 665, 211 Cal. Rptr. 368 (Cal. Sup. Ct., Feb. 28, 1985) *(The Citation*, August 1, 1985, Vol. 51, No. 8)

UPDATE: LAW LIMITING DAMAGES

On October 15, 1985, the U.S. Supreme Court allowed California to impose a $250,000 limit on the amount a victim of medical malpractice may recover for such noneconomic losses as pain and suffering. A California man who suffered a heart attack said the limit denied him adequate compensation for a late diagnosis that could shorten his life. *(Trentonian*, October 16, 1985)

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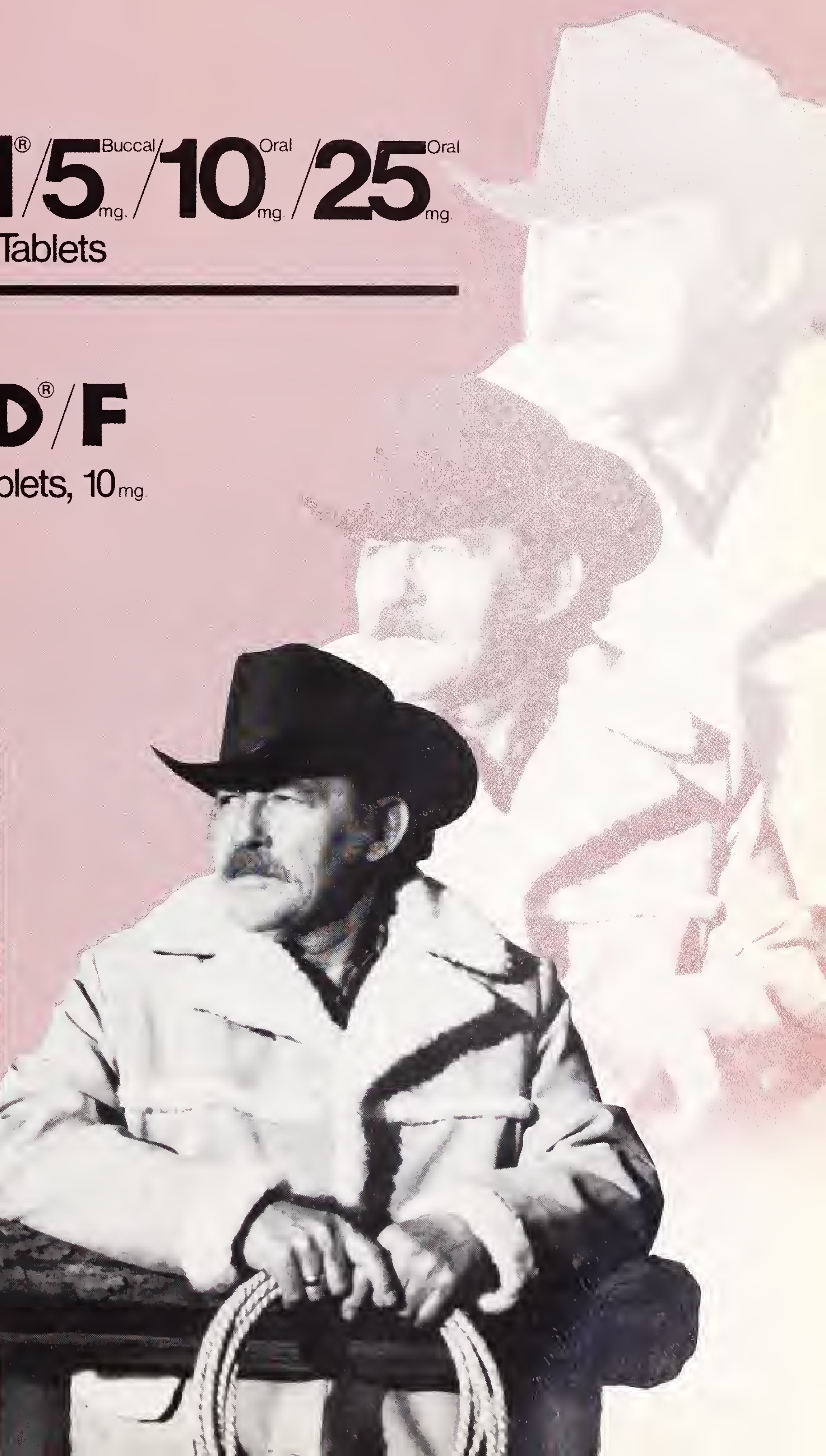

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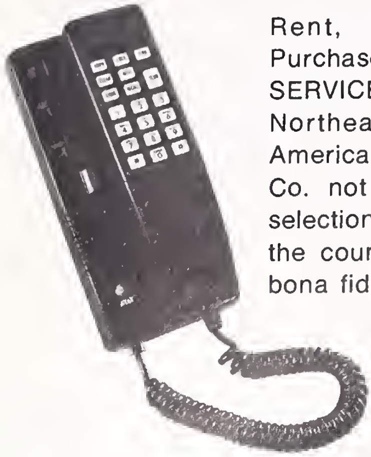

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EDITORIAL

Task Force Coalition with Senior Citizens

Who is a better ombudsman for elderly New Jersey residents than the New Jersey physician?

Ralph J. Fioretti, M.D., President of the Medical Society of New Jersey said, "We must show senior citizens that they will be the victims of this cost containment (through PRO legislation) and that we, their doctors, whom they hold in high esteem, stand ready and willing to fight for their rights. We must show them that we care and we will not let them down. We must be guided by conscience to do what is right. To accomplish this end, I am announcing, therefore, that my first action as President will be to appoint a task force of physicians. I will charge it with the responsibility of meeting with senior citizens around the state, to explain what is happening to them through government action and to discuss the pitfalls raised by Medicare cutback and underutilization."

A. Ralph Kristeller, M.D., Chairman of the Task Force Coalition with Senior Citizens wrote, "The purpose of the Coalition is to promote constructive dialogue between physicians and senior citizens in order to identify areas of agreement, areas of disagreement, and above all to try to prevent misunderstanding. Its further purpose is for reasonable and concerned citizens to influence health care policy in a positive way."

Anne R. Somers, Professor, commented, "Perhaps the most encouraging aspect of the current scene is the rapid growth of professional interest in geriatrics, in the changing health needs of the elderly and in the need to adjust our health care institutions to meet the changing needs. If the current inadequacies of health care for the elderly are not to be made worse, rather than better, by changing national policies, it will require the work of many people."

The Task Force has begun to meet with senior citizens to carry forward its mandate. Here are some of their quotes, which explicitly express some of their complaints:

1. Sending a bill for saying "hi" through the doorway of the hospital room.
2. Wait three weeks for an appointment, sit in the waiting room for three hours, and he sees me for five minutes ("3-3-5 syndrome").
3. More acceptance of assignment for the needy.
4. No written directions for medications.
5. Fees are too high.
6. Too many referrals.
7. Doctors are taking advantage of Medicare.
8. Does not give me his full attention.
9. More attention to and earlier arrangements for hospital discharges.
10. Pencil and paper at the hospital bedside which can be used as a diary and reminders.
11. Emergency rooms need to study the problem of waiting time. Only if you come in by ambulance do they see you right away.
12. Dispenses too many pills at one time.
13. Receive bills from unknown physicians in the hospital.
14. Why do I need two medical doctors?

Is there any physician reading this editorial whose patients are not saying some of those things? I doubt it!

Is there any physician reading this editorial who does not know what his duties to the elderly are? Unlikely!

No task force coalition can do the whole job by holding meetings with senior citizens around the state of New Jersey. The elderly have more medical problems, less income, and more medical expenses than any other group. Many (5 percent) are institutionalized and half of them have had less than two years of high school education. Almost all of the senior Americans meet up with a physician at some time or another.

Who is a better ombudsman for elderly New Jersey residents than a New Jersey physician? That rhetorical question can have only one answer. Anne R. Somers is right: "If the current inadequacies of health care for the elderly are not to be made worse, rather than better, by changing national policies, it will require the work of many people."

Talk to your elderly patients about their problems. Let them express their feelings to you and listen to them! In simple terms explain what you can. In short, appoint yourself as an unofficial member of the Task Force Coalition with Senior Citizens. A.K.

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MEDICAL SOCIETY OF NEW JERSEY—220TH ANNUAL MEETING

HOSPITAL GOVERNING BOARDS*

Conflict of Interest in Certificate of Need Regulation

VINCENT A. MARESSA, J.D.

MSNJ has called for the repeal of the certificate of need law in New Jersey.

New Jersey maintains one of the more extensive certificate of need programs in the country. Currently, the Department of Health, with the support of the University of Medicine and Dentistry, is attempting to extend the process to include physicians in private practice. A bill (S-2869) introduced at their request is pending in the State Senate.

The proponents center their campaign on several key issues. They assert: the certificate of need process is cost effective, it will produce well-managed hospital monopolies, and, by including doctors, all the major providers will be playing on a level field.

Their assertions are absolutely unfounded. The certificate of need is not cost effective. Major university studies have found it to be ineffective, cumbersome, and expensive. The franchising of monopolies via the certificate clearly has not produced a well-managed, efficient monopoly. Sweeping doctors into the net will not produce a level field. Doctors are at risk. Hospitals are not. Hospitals receive guaranteed rates for indigent care under the DRG program. Doctors receive no factor for indigent care. Doctors pay state and federal taxes. With the exception of the one proprietary hospital in New Jersey, hospitals do not pay taxes. Hospitals are multimillion-dollar conglomerates. The average medical practice has three physician members. Hospitals have hundreds of employees. Doctors have less than five.

All other factors aside, the certificate of need produces a licensed monopoly, and the process itself is prone to an incredible amount of manipulation. Conflicts of interest are rampant and pervade the entire structure. Close scrutiny produces startling revelations.

A major influence in the system is the State Health Coordinating Council. It is appointed by the governor and consists of a number of people from various sectors of society who review all applications and then make recommendations to the State Commissioner of Health, who then makes the decision to grant or deny a certificate.

Hospitals under the direct control of the Department of Human Services are exempt from the process. The largest operator of acute hospitals in New Jersey is the state itself, through UMDNJ by virtue of its directly managed and affiliated hospitals.

Given the size and dispersion of that hospital network, it is difficult to imagine a certificate of need issue that does not favorably or adversely impact on the UMDNJ system and/or interests. The President of the University sits on the State Health Coordinating Council. He has an interest—pro or against—in virtually every application presented. The fact that he may abstain from a given vote in no way diminishes his capacity to influence the other members of the State Health Coordinating Council on the issues before them by both public and private discussion. He is the chief operating officer of the University and the operator of the state-run acute care hospitals. The president of UMDNJ is a close advisor to the Commissioner.

The Commissioner of Health, likewise, is in a severely compromised position. As the final decision-maker, he is the keystone. He also is a member of the Board of Trustees of UMDNJ. It strains credulity to accept the premise that he can function objectively in deciding certificates that have any impact on UMDNJ.

I am in no way criticizing either man; I am simply pointing out that the system within which they are functioning is fatally flawed. It is wrong for any system to demand that people deny their humanity. The answer to this dilemma is to repeal the system. It has not worked. It will not work. It is subject to manipulation.

CONCLUSION

The Medical Society of New Jersey has called for repeal of the certificate of need law in New Jersey. Certain hospital administrators are in support of the Medical Society of New Jersey position. The New Jersey Hospital Association has not declared a position. It is time it did. The Medical Society of New Jersey cannot sustain an effort to help hospitals when their Association remains indifferent and passive. Every hospital in New Jersey has the obligation to form a position either favoring or repealing the certificate of need law in this state.

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*The material for this column is coedited by Arthur Krosnick, M.D., Editor, *NEW JERSEY MEDICINE*, MSNJ; Vincent A. Maressa, J.D., Executive Director, MSNJ; and James E. George, M.D., J.D., Director of Professional Liability, MSNJ.

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OPINION

ARE THE PRIVATE PRACTITIONER AND FEE-FOR-SERVICE

ENDANGERED SPECIES?

Those old enough to have lived through the Depression of the 1930s remember the confusing alphabet soup of President Roosevelt's federal agencies—NRA, WPA, and CCC. Today's health care scene presents both physicians and patients with a new set of letters: HMO, IPA, and DRG. It would be of some value, I believe, to analyze what HMO (Health Maintenance Organization) and IPA (Individual Practice Association) mean to the private practicing physician.

The number of physicians has been increasing yearly and it seems the trend will continue throughout the 1980s. Couple this with the economic squeeze on hospitals and physicians, and you have severe competitive

Howard D. Slobodien, M.D.

threats for those of us who have believed in the traditional one-to-one physician-patient relationship, where care is provided in the physician's office and the physician is reimbursed on the basis of services rendered.

In addition, hospital costs for inpatient services will continue to escalate as less costly procedures are treated on an ambulatory basis, leaving the hospitals with a higher and higher percentage of the more serious medical and surgical problems. This has been one of the factors that already has stimulated many hospitals to undergo corporate reorganization and to set up for-profit divisions, many of which compete directly with the private practitioner.

One of the few viable answers, under present FTC constraints, is the IPA. But IPA/HMOs come in different forms. Some use capitation fees and some negotiate fees with individual physicians or groups. But only one type allows the continuation of true fee-for-service. In this type of HMO, the physician is represented by an organization composed solely of local physicians. The organization, with its boards and committees, provides the physician member with a voice in varied issues, including reimbursement, standards of medical care, and peer review. This, I feel, is an essential element for physician satisfaction, which does not exist in other types of IPA/HMOs.

In this IPA/HMO model, the patient selects a participating primary care physician, who then supervises and manages the patient's health care needs, treating personally, if possible, and referring to nonprimary care physicians as indicated. Under this system nothing is changed in the delivery system but the way it is financed, and there are no financial incentives to keep costs down. I have some concern about those capitation plan IPA/HMOs in which the physician is assigned a number of patients for whom he is paid, whether he treats them or not; I am concerned that needed primary care visits and recommended consultations may be decreased. I have real worries about those capitation plans that reward the primary care physician with bonuses for restricting hospitalizations and referrals, even though the average physician can be expected to practice in a moral and ethical manner for the good of the patient.

The fee-for-service IPA/HMO may be the last remaining way for physicians to have some measure of control over their services. The patient's needs are prepaid, except for minimal copayments. The office physician-patient relationship is maintained. The physician is paid a reasonable fee for his services. Paperwork is kept to a minimum. Reimbursements, standards of care, and review procedures are determined by practicing physicians. Office and other out-of-hospital procedures are encouraged and reimbursed. So, this type of plan can serve the patient's needs, conserve costs to industry, and help to satisfy government's demands to control health care costs.

In the turbulent years ahead, it may be one of the few remaining options left to keep the private practice physician from becoming a nostalgic American anachronism.

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BIOSOCIAL NEEDS OF CHILDREN ADMITTED TO AN URBAN HOSPITAL

SOPHIE PIEROG, M.D., AUSTIN C. HILL, PH.D., RONI BROOKS, R.N., ISMAEL MANASRA, M.D., GERALDINE JOURNITZ, R.N., JERSEY CITY*

In a month of admissions at an urban hospital, there was evidence of an overwhelming preponderance of children and families having psychosocial problems that challenge the primary care physician. It is difficult to make the appropriate authorities expend funds to help solve these problems.

In 1978, Van Gelder, in his address to the American Academy of Pediatrics (AAP), noted that primary care was "fashionable" and that this included consideration of a child's and family's lifestyle, education, and support services available to them.[1] This also has been brought into focus by the AAP concerned with the training of young pediatricians; residency program curricula are to include the recognition and management of the psychosocial or biosocial problems of children and families.[2]

In a 1979 study reported by nine pediatricians representing various types of pediatric practices, Goldberg et al.[3] found the incidence of mental health problems in children (i.e. adaptation reaction, learning disorders, conduct disorders) to be 1.4 to 7.8 percent of their patients. These mental health problems were highly associated with some factors which are considered biosocial. Examples include the families' health insurance source (most of the families were on Medicaid) and the absence of the father.

We feel that the incidence quoted by Goldberg and his associates is actually much higher but, as Starfield noted, there is a dearth of descriptive statistics to show what proportion of a pediatric practice these concerns may involve.[4]

One of the problems in attending to the biosocial needs of the pediatric patient and the family may be the lack of sufficient funds provided in reimbursement rates by third-party payment health insurance groups. Hospitals with pediatric services and training programs as well as private practitioners are involved. Funds are limited and hospital administrators, concerned with Diagnostic Related Groups (DRG), third-party reimbursements, peer review groups, and lengths of hospital stay, may consider that meeting the biosocial needs of a child and the family, providing the personnel trained to teach residents, and working with families as not reimbursable and, therefore, a financial burden to the hospital.

To document the biosocial/developmental problems that impact on their present and future health needs and those of their families, we studied a group of children admitted to one unit of a pediatric service for a one-month period.

METHODS

Jersey City had 223,532 citizens in 1980, most of whom are blue collar workers; a number of citizens are on public assistance. Recently, with national economic setbacks, many family members became newly unemployed; 20 percent are at the poverty level.[5] New immigrants (both legal and illegal) also have arrived in the community.

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The pediatric department at the Jersey City Medical Center, which is considered the only hospital in Hudson County as a referral center, receives the critically ill children and neonates from five private hospitals in the county. There were 3,675 patients admitted to pediatrics in 1982; the delivery rate was 2,824 (second largest in the state). The neonatal service also has an intensive care nursery, with a county-wide neonatal transport service.

In a single day, the pediatric and neonatal census is one-third the hospital population (average of 380 to 425 total patients per day). The social service department has a staff of ten yet only one is assigned to the pediatric in-and-out patient area, the neonatal services including the neonatal intensive care unit, and to the obstetrical service which has a very large population of high-risk mothers.

One month was selected at random, May 1983, and children admitted to the 5-North, older children, and adolescent services (age range of 3.6 to 15.11 years of age) were studied.

An analysis was made of medical problems on admission. The child's illness, past history of illness and/or developmental delays, family ilnesses, and the source of their medical insurance also were investigated. A simple questionnaire was devised for the resident and nursing staff to document some biosocial problems of the child and family including mother's age at first conception, marital status, degree of indigency (welfare), family household numbers, and any evidence of family violence, behavior and school problem of the child.

RESULTS

One hundred children were admitted to this unit from May 1, 1983, to May 31, 1983; 52 were male and 48 were female. Of these 100 children, 57 were black, 22 were Hispanic-surnamed, 10 were white, and 11 were Vietnamese, Indian, white/black, or Chinese. We analyzed the admissions as follows:

1) Children who were admitted for an acute illness which would not have long-lasting effects on the child's future adaptation and those having no behavioral or developmental problems on parent/child interview were placed in Category 1. These children then would be considered as a "nonrisk" group—medically, developmentally, and behaviorally.

2) The children who had no history of behavioral or developmental problems but who were admitted because of a medical/psychological illness which could have serious long-term effects on the child's future adaptation were placed in Category 2.

3) The remainder of the children who had a history of chronic medical, psychological, behavioral, or developmental problems and who were admitted because of their chronic illness or an acute intercurrent illness were placed in Category 3.

The families of the children in each of these categories were analyzed for biosocial problems that could affect the child's future well-being (Table).

Thirty-four children (34 percent) were in the nonrisk Category 1; 8 children (8 percent) were in Category 2; and 58 (58 percent) were in Category 3. This means that 58 percent of our admissions within one month were children whose lives were already compromised by medical illness, behavioral, and/or developmental problems. An additional 8 percent were admitted with illnesses which would have a definite impact on their future lives.

Of the 100 children admitted during this period, the families of 74 were considered medically indigent—on welfare and on Medicaid for health care; 14 families had no medical insurance. Single mothers headed 73 of the 100 families and of these 73, the mother's whereabouts were unknown in 2; the grandmother was the caretaker in 1 family, and 1 child was in foster care. Thirty-eight mothers of these families were under 18 years of age at first conception. These mothers had a total of 162 children in the household; this is a ratio of 4.3 children for every 1 mother. We did not know the age status at first conception in 7 mothers of the 100 children admitted; these 7 mothers had a total of 23 children. The remaining 55 mothers who were not teenagers at first conception, had a total of 132 children or a ratio of 1 to 2.4 or 2.4 children per mother.

There was a history of a major medical illness in the family—in mother, father, or siblings—in 58 of these households: hypertension, chronic asthma, major heart disease, sickle cell disease, renal disease, or epilepsy.

Even in the group of 34 children in Category 1 (nonrisk group) there were a number of biosocial factors that could impact adversely on the child's future health and development. Indigency or dependence on welfare and Medicaid was found in 21 of 34 (61.7 percent) and 5 families had no insurance; 21 (62 percent) were single-parent households with 13 mothers who were teenagers with their first pregnancy. The major caretaker and/or the siblings in 17 (50 percent) of these families had major or chronic illnesses.

The eight children in Category 2 (whose illness at this admission would impact on their future health and/or potential) also had family problems. Six of eight families were on welfare/Medicaid and one family had no insurance; seven families were headed by single mothers and five had major illnesses in family members.

In 58 of the Category 3 families (those whose children were admitted for known chronic organic or psychological disease), 47 were welfare/Medicaid recipients and 8 had no medical insurance. Single mothers headed 45 of these families and 25 (43 percent) were teenage mothers at first conception.

DISCUSSION

Children's health and their growth and development to maturity is absolutely affected by biosocial problems. A family with no medical insurance usually would not seek preventive health services;[6] their medical contacts would primarily be for acute illnesses. A family on welfare or public assistance frequently lives in unhealthy or unsafe housing areas and may not be able to budget for appropriate or nutritious food for the family.

A single parent, coping with indigency, a serious illness, as well as with a number of dependent children, may be too depressed to attend to a child's health or developmental problems. Male role models are extremely important in a child's development; transient boyfriends and stepfathers may not meet this need.

TABLE

Analysis of Biosocial Data on 100 Children Admitted to Pediatric Unit

	Category 1	Category 2	Category 3	Total
Total No. of Children	**34**	**8**	**58**	**100**
Ethnic Group				
• Black	21	5	31	57
• White	1	–	9	10
• Hispanic-surnamed	7	2	13	22
• Others	5	1	5	11
Welfare/Medicaid	21	6	47	74
No insurance	5	1	8	14
Single mothers	21	7	45	73
Mothers 18 years at first conception	13	–	25	38
Total no. of children born to these mothers	43	–	119	162
Age at first conception not known	2	–	5	7
Total no. of children born to these mothers	9	–	14	23
Major illness in family	17	5	36	58

Teenagers who become mothers usually are school dropouts and candidates for public assistance; their lack of understanding of child care practices and child behavior definitely can influence the development and the behavior of the young child. Depressed, they may go on to have large families, and thus compound their problems.

One would expect that an urban hospital would abound in biosocial problems of many of the children admitted for medical care. Our study corroborates this expectation. This, however, does not ease the burden of actually coping with it by the medical staff. As the literature indicates, efforts to attend to these problems require more people who spend time and effort on an almost one-on-one basis, to work with these families.

At this hospital with definite evidence of great family needs, one-third of the total patient population of the hospital (pediatric, obstetric, and nursery services) utilize the services of one-tenth of the social service department. There is a wealth of experience for the pediatric residents to attend to total child health care. But even this wealth of experience suffers from lack of faculty and social services to teach them to recognize these problems and deal with the families who are in need. Hospital administration is not reimbursed for extra faculty or social service or child-life services staff to attend to biosocial needs of the hospitalized child, much less for staff to follow this child and family in their community.

Though great advancement has been made in technologic skills in treating the child for a variety of complex medical illnesses, the health needs of children and adolescents are much broader in scope. As more fragile patients survive because of our technical skills, we also will begin to have a pool of children with residual disabilities, who are even more susceptible to the effects of environmental and social stresses than the general population. With increasing divorce rates, greater numbers of single-parent families, family violence, alcoholism, and substance abuse and with economic setbacks and decrease of funds allotted to health needs, the child and the family may suffer any number of stresses that impact on their health.

CONCLUSION

This study analyzed only a minimal number of factors considered biosocial impacting on children's health care; however, it verifies what pediatricians have known and what they have been coping with in the urban hospital. Starfield[4] stated that there was a lack of descriptive statistics of a pediatric practice to show the proportion of biosocial and medical problems which need attention in the total care of the child and family. We have tried to report some statistics of our hospital practices to show how prevalent and overwhelming these problems can be.

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REVIEW ARTICLE: METABOLIC MYOPATHIES

MARVIN I. RUDERMAN, M.D., AND GEORGE ZITO, M.D., NEWARK*

An increased number of conditions involving muscle metabolism (glycogen, lipid, and mitochondria) are recognized in children and adults. They may produce progressive weakness or myoglobinuria. Effective treatment is evolving for some of these disorders.

Disorders of muscle which can be attributed to specific metabolic defects have been recognized in greater numbers expanding our understanding of these conditions and, in some cases, leading to effective treatment.[1-7] Still other less well-defined disorders are known to involve certain aspects of muscle metabolism while the specific cellular defect remains unknown.[8-10] The insight gained from this elucidation of these previously undiagnosed myopathies (sometimes labeled limb girdle syndromes) also has increased our knowledge of normal muscle physiology. This review will discuss the metabolic disorders of glycogen, lipid, and mitochondria which affect muscle (Figure).

ACID MALTASE DEFICIENCY

Muscle diseases of glycogen and lipid metabolism can be considered clinically to produce one of two basic syndromes: progressive muscle weakness or exercise intolerance and myoglobinuria. The first such condition causing rapidly progressive muscle weakness was identified in 1932 by Pompe who described the infantile form of acid maltase deficiency.[11] These infants are normal at birth, but develop hypotonia, weakness, cardiomegaly, heart failure, and mild hepatomegaly within the first few months of life. There often is tongue enlargement. Death, due to cardiopulmonary failure, usually occurs by age two. A childhood form of acid maltase deficiency exists in which patients are normal in infancy, often exhibit delayed walking, and suffer from a proximal limb-girdle weakness. There is no cardiomegaly and hepatomegaly is less frequent than in the infantile form. Skeletal muscle weakness is progressive and usually leads to respiratory failure and death before age 25. Onset of Pompe's disease in the third and fourth decade (but sometimes as late as the sixth decade) without cardiac or hepatic involvement, also is well-described as the adult form of acid maltase deficiency. In adults, the clinical syndrome is that of a progressive myopathy often with prominent respiratory compromise.

Inheritance of acid maltase deficiency is autosomal recessive and prenatal detection by amniocentesis is possible. Laboratory studies reveal a variably increased creatine kinase (CK) (2 to 12 times normal), electromyography (EMG) with myopathic features as well as fibrillations, positive sharp waves, myotonia (without clinical myotonia), and a reduced urinary excretion of acid maltase.[1,13,15] Periodic acid-Schiff (PAS) positive vacuoles (and a positive acid phosphatase reaction) are prominent in muscle biopsies from infantile cases, but less conspicuous or absent in adults. Biochemically, acid maltase (alpha 1-4 glucosidase) is reduced or absent in skeletal muscle, heart, brain, and urine. Late-onset cases have a 3 to 12 percent residual acid maltase activity, but the basis for the three distinct clinical forms remains unclear. There is no effective treatment at this time.

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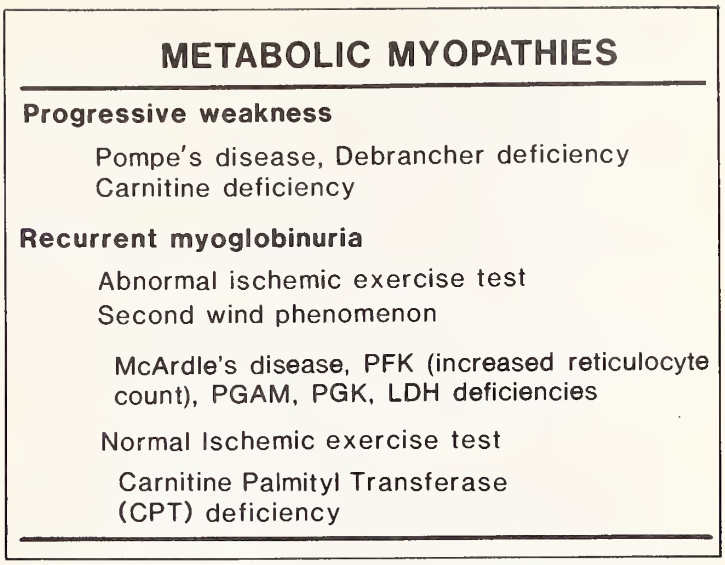
DEBRANCHER DEFICIENCY

Debrancher deficiency most often is a self-limited process of hepatomegaly, growth retardation, and fasting hypoglycemia in childhood, without myopathy. It has been considered only rarely to cause diffuse muscle weakness in adolescent or adult life, but eight recent cases illustrated adult-onset slowly progressive myopathy with proximal and distal weakness.[17,18] Resting CK usually is markedly elevated. The muscle biopsy reveals a vacuolar myopathy with accumulation of glycogen, and the deficiency can be demonstrated on biochemistry of muscle tissue. One study reported clinical improvement with a high-protein diet in a seven-year-old boy.[19]

CARNITINE DEFICIENCY

The discovery of carnitine deficiency in 1973 as a lipid storage disorder causing progressive myopathy has led to effective treatment of this condition and elucidation of muscle and systemic forms.[5,6,20,21] Carnitine acts as a carrier for medium and long-chain fatty acids facilitating their transfer across the mitochondrial membrane for beta oxidation. It is believed to be synthesized in the liver and then transported to blood and muscle.[5] The clinical features of the myopathic form of carnitine deficiency consist of proximal limb and trunk muscle weakness usually beginning in childhood. It presents as a limb girdle syndrome. The first reported case initially was believed to be polymyositis until the muscle pathology was examined.[20] Facial and pharyngeal weakness may be present. The CK is moderately increased, a myopathic EMG pattern is seen, and the serum carnitine measurements are normal or only slightly decreased. On muscle biopsy, a severe vacuolar myopathy is noted, with vacuoles staining positive for lipid collections with the oil red O stain. Biochemically, the muscle carnitine is reduced to 5 to 32 percent of controls. Treatment with oral dl-carnitine or steriods is effective in most patients with significant improvement in strength.[1,5,6,20,22] Although uncertain at present, it is thought that the myopathic form of the carnitine deficiency results from the inability to transport carnitine into the muscle.

A systemic form of carnitine deficiency was described in 1975.[21] In these cases, serum carnitine is decreased uniformly, and low values for muscle, liver, and heart carnitine are found. The serum CK is variably increased, EMG is myopathic, and muscle biopsy findings are identical to those of muscle carnitine deficiency. Clinically, progressive proximal limb and trunk muscle weakness beginning in infancy or early childhood is accompanied by episodes of hepatic encephalopathy resembling Reye's syndrome. Patients develop nausea, vomiting, confusion, coma, hepatomegaly, hypoglycemia, hyperammonemia, elevated liver function tests, and lactic acidosis. This condition is believed to reflect a defect in hepatic biosynthesis of carnitine.[1,5,23]



METABOLIC MYOPATHIES

Progressive weakness

Pompe's disease, Debrancher deficiency
Carnitine deficiency

Recurrent myoglobinuria

Abnormal ischemic exercise test
Second wind phenomenon

McArdle's disease, PFK (increased reticulocyte count), PGAM, PGK, LDH deficiencies

Normal ischemic exercise test

Carnitine Palmityl Transferase (CPT) deficiency

Figure—Classification of glycogen and lipid disorders of muscle based upon clinical features and ischemic exercise test. Debrancher deficiency also is associated with an abnormal ischemic exercise test. PFK indicates phosphofructokinase; PGAM, phosphoglycerate mutase; LDH, lactate dehydrogenase.

Treatment with oral carnitine has been effective in 75 percent of patients studied with the myopathic form, and 66 percent of those with the systemic form. Prednisone can be used with 60 percent efficacy in the myopathic form if results with carnitine are unsatisfactory.[5] Prednisone was effective in reducing intracellular lipid in the myopathic form when sequential muscle biopsies were studied, but carnitine concentration did not change.[24] One case of systemic carnitine deficiency studied showed improvement with oral carnitine therapy, but no rise in liver or muscle carnitine despite normalization of serum carnitine.[21] In another instance of systemic carnitine deficiency in a 20-year-old woman with episodes of Reye's encephalopathy and progressive muscle weakness, clinical response to oral L-carnitine was accompanied by a rise in serum and muscle carnitine content.[23] Other cases of carnitine deficiency share some of the features of one of these forms, but with laboratory results more consistent with the other type. Examples are cases with the clinical picture of systematic carnitine deficiency but with normal serum carnitine, and others with muscle weakness but no liver disease and consistently low levels of serum carnitine. Other potential causes of secondary carnitine deficiency are malnutrition with liver disease and sepsis.[5]

Patients have been described with variable degrees of weakness in association with lipid storage in muscle not due to carnitine deficiency. A syndrome of congenital ichthyosis, myopathy, and lipid storage in muscle, liver, skin, leukocytes, and gastric mucosa has been recognized.[8,25] Muscle carnitine is normal in these cases.

MITOCHONDRIAL MYOPATHIES

Disorders of muscle mitochondria, often accompanied clinically with progressive weakness, have been identified and classified based on muscle biopsy findings of morphological disruption of mitochondria. In the majority of these cases, no specific biochemical defect yet has been identified. The Kearns-Sayre syndrome (also called ophthalmoplegia plus, oculocraniosomatic dystrophy, and ragged red fiber disease) is a multisystem disorder characterized by progressive external ophthalmoplegia, atypical retinitis pigmentosa, cardiac conduction block, sensorineural hearing loss, mental retardation, proximal muscle weakness, and cerebellar dysfunction. Increased serum lactate is found at rest in some patients, and cerebrospinal fluid (CSF) protein is elevated. Ragged red fibers on muscle biopsy with trichrome stain represent disorganized mitochondria and also are seen on the electron microscope.[10,26] Similar evidence of morphologic abnormalities of muscle has been observed in a host of other conditions including certain congenital slowly progressive myopathies, and isolated cases of facioscapulohumeral syndrome, myopathy with growth retardation, lactic acidosis, and various cerebral symptoms.[5]

A defect of uncoupling of oxidative phosphorylation (Luft's disease) was described in 1962 in two patients with a hypermetabolic state, normal thyroid function tests, and mild generalized weakness.[9] A third reported case was noted to improve with the use of chloramphenicol, presumably due to a depressant effect on mitochondrial respiration.[27] Among the recognized defects of the mitochondrial respiratory chain, cytochrome-c-oxidase deficiency has been described in several patients with a fatal infantile myopathy, renal dysfunction, and lactic acidosis, as well as a benign myopathy in infancy which improves.[28,29]

MYOPHOSPHORYLASE DEFICIENCY

The clinical syndrome of exercise intolerance and myoglobinuria is seen in another group of glycogen and lipid storage disorders, several of which have been elucidated in the last few years. McArdle's disease (myophosphorylase deficiency) first was described in 1951 in a 30-year-old man with a history of muscle pain with exercise.[30] The symptoms resolved upon resting. A true contracture (electrically silent) developed with ischemic exercise. Absence of the normal rise in blood lactate during ischemic exercise of a limb was recognized by McArdle, and has remained a useful screening test for this disorder, as well as phosphofructokinase deficiency (PFK), phosphoglycerate mutase (PGAM), and muscle lactate dehydrogenase (LDH) deficiencies which are discussed below.[3,4,30-32]

The onset of symptoms in myophosphorylase deficiency usually is late in childhood or adolescence, but other forms in infancy and later life have been described.[1,33-35] Brief, strenuous exercise or sustained activities such as swimming or jogging tend to induce symptoms of muscle pain, stiffness which may progress to muscle cramps, and myoglobinuria if the exercise is continued. The myoglobinuria may lead to acute renal failure. More moderate exercise, e.g. walking, is better tolerated, and often a second wind phenomenon is noted by patients, in which symptoms of muscle pain and stiffness can be overcome if the pace of activity is reduced briefly before resuming the prior level. This is thought to be related to mobilization of free fatty acids and increased blood flow to muscles.[1,15,32] A small minority of patients may develop progressive weakness with time. A recent case was successfully treated with a high-protein diet resulting in improved endurance.[36]

Laboratory investigations in McArdle's disease show the resting serum CK to be variably increased. On muscle biopsy, subsarcolemmal PAS-positive vacuoles containing glycogen usually are seen at the periphery of fibers, but may be absent in the late-onset types.[34] Histochemically, phosphorylase activity is absent, except in vessel walls and regenerating fibers.[1,37,38] Biochemically, there is absence or reduction of phosphorylase activity, limited to skeletal muscle. The defect is in the M subunit; heart and liver have isoenzymes. Of interest is that fetal muscle contains an isoenzyme retaining phosphorylase activity.[1] Although there is a male predominance, McArdle's disease is believed to be inherited as autosomal recessive. Treatment at present generally is limited to restriction of exercise. The patient's development of muscle pain usually provides the necessary warning to prevent inducement of myoglobinuria.

PHOSPHOFRUCTOKINASE DEFICIENCY

Phosphofructokinase deficiency (PKF) presents a similar clinical picture to McArdle's disease, though in some patients nausea and vomiting may accompany the exercise-induced muscle pain, stiffness, and subsequent myoglobinuria.[1] A late-onset form with progressive weakness also has been described.[39] There is a failure of ischemic exercise to induce a rise in the serum lactate. Several clinical laboratory clues may suggest the diagnosis of PFK deficiency: elevated reticulocyte count, increased serum bilirubin level and uric acid in some patients. A partial defect in erythrocyte phosphofructokinase, which shares a subunit in common with muscle PFK, is believed responsible for the hemolysis found in these cases.[32] The diagnosis is established by muscle biopsy where sarcolemmal deposits of glycogen, or glycogen vacuoles, usually are identified. A histochemical stain for phosphofructokinase is available for the identification of the enzyme defect.[40] Severe reduction in muscle PFK is found biochemically. Specific therapy for this condition is not available.

OTHER DISORDERS OF GLYCOGEN METABOLISM

In the past several years, three other enzymatic defects of glycolysis have been recognized as responsible for a clinical syndrome of exercise-induced pain and myoglobinuria.[3,4,41,42] In these disorders, some rise (but usually less than normal) in lactate during ischemic exercise may occur, and glycogen accumulation is not seen on light microscopic examination of muscle biopsy. Muscle lactate dehydrogenase (LDH) deficiency was identified in 1980 in a patient experiencing recurrent myoglobinuria after intense exercise.[7] The presence of an LDH isoenzyme in muscle (LDH-H4) was responsible for the small amount of residual LDH activity found. Phosphoglycerate kinase deficiency was described in a 14-year-old boy with a history of exercise-induced myalgia and weakness since age 5, and three episodes of myoglobinuria following exercise, at ages 13, 14, and 15.[3] Between attacks, the serum CK was normal and electromyography was normal. An ischemic exercise test caused prolonged contracture, with no rise in lactate. On muscle biopsy, there was a slight increase in intensity of PAS stain for glycogen, but no focal accumulations. Biochemically, the glycogen content was normal, but PGK activity was found to be 5 percent of normal mean. A partial defect of this enzyme was found in the patient's mother, who was clinically normal. The presence of residual enzyme activity in the cases of PGK and PGAM deficiency appears to explain the absence of glycogen accumulation, and normal light microscopy muscle morphology.[3]

Phosphoglycerate mutase (PGAM) deficiency was described in 1982 in a 52-year-old man with a history of exercise-induced cramps and pigmenturia since adolescence.[4] Two other reports involved a 17-year-old girl and a 24-year-old man with similar symptoms since age 13.[41,42] Forearm ischemic exercise in the first two cases resulted in a subnormal rise in venous lactate, but a normal rise in the third case. Muscle biopsy in the first case revealed only a slight increase in the PAS stain for glycogen, but was more pronounced in the second patient. In both instances, the electron microscope demonstrated abnormal accumulation of glycogen particles between myofibrils and subsarcolemma. Biochemically, glycogen content in muscle was at the upper limits of normal in the first case, moderately increased from the second patient, while normal in her parents. PGAM activity was 3.6 percent and 6 percent of the normal mean in the first two cases, respectively. Residual enzyme activity was due to the presence of the BB PGAM isoenzyme in muscle. A defect of the M subunit of this enzyme is suggested, and the second case, in which both parents exhibited approximately half the normal PGAM activity in muscle biopsies, indicates autosomal recessive transmission.[41]

CARNITINE PALMITYL TRANSFERASE DEFICIENCY

Carnitine palmityl transferase (CPT) deficiency, first described in 1973, represents an important and perhaps the most common identified cause of hereditary exercise-induced myoglobinuria.[2,5] This enzyme is responsible for the movement of long-chain fatty acids across the mitochondrial membrane, by esterification with carnitine and subsequent release from carnitine to enter beta oxidation. Resting muscle primarily utilizes lipids; however upon heavy exercise, particularly for short periods, glycogen is utilized for muscle energy. During prolonged exercise, there is a shift back to lipid metabolism. During starvation, muscle energy primarily is derived from lipids.

The relationship of diet and exercise particularly is relevant to the clinical syndrome seen in CPT deficiency. Symptoms usually begin in childhood, consisting of muscle pains and myoglobinuria most often precipitated by prolonged exercise, i.e. several hours of hikes, athletic games, fasting. Short-duration exercise is well-tolerated. Although muscle pain develops after exercise, cramps are not found, and insufficient warning often results in myoglobinuria. The ischemic exercise test is normal; resting CK and EMG usually are normal. Between attacks of myoglobinuria, light microscopic muscle biopsies were normal in two-thirds of the patients. Biochemically, CPT activity has been found to vary between 5 and 24 percent of the normal mean.[5] The condition is familial but it is not clear whether inheritance is autosomal recessive or X-linked. Treatment with a high-carbohydrate, low-fat diet reduces the frequency of attacks and improves exercise tolerance in most patients. Caution also is given against fasting.

> ***Treatment is available in carnitine deficiency and we hope it will be available in other metabolic muscle disorders soon.***

EVALUATION OF METABOLIC MYOPATHIES

The evaluation of patients with suspected metabolic myopathies initially will include a careful history and examination and serum enzyme determinations (CK, aldolase). Among the disorders associated with progressive weakness, cases of infantile Pompe's disease will be suspected by the associated cardiac and hepatic disease. Older patients with acid maltase deficiency have a predilection for respiratory compromise (often out of proportion to weakness). Adults present with a limb girdle syndrome as seen with carnitine deficiency as well. Cases of systemic carnitine deficiency are recognized by the coincident recurrent hepatic failure. Electromyography usually will identify myopathic potentials, and may show abundant spontaneous activity, particularly in Pompe's disease. Muscle biopsy is necessary to establish the diagnosis, and should include histochemical as well as biochemical studies. A history of recurrent myoglobinuria merits the performance of an ischemic exercise test.[31] This will be abnormal in cases of phosphorylase, phosphofructokinase deficiencies, and show a partial (but usually subnormal) rise in lactate with the newly described defects in glycolysis. The test is normal in carnitine palmityl transferase deficiency. In these cases, light microscopic muscle biopsy studies may be normal, or nondiagnostic, while electronmicroscopy usually identifies abnormal lipid and glycogen deposits. Biochemical studies are essential to the correct identification of the enzyme defect. The information provided by this analysis can provide important practical guidelines for the patient, as well as prognostic and genetic information.

Although the metabolic myopathies discussed in this review are uncommon, they are well-defined clinical disorders caused by defects in the normal cellular function of the muscle. Effective treatment now is available in carnitine deficiency and we hope it will be available in other metabolic muscle disorders in the future.

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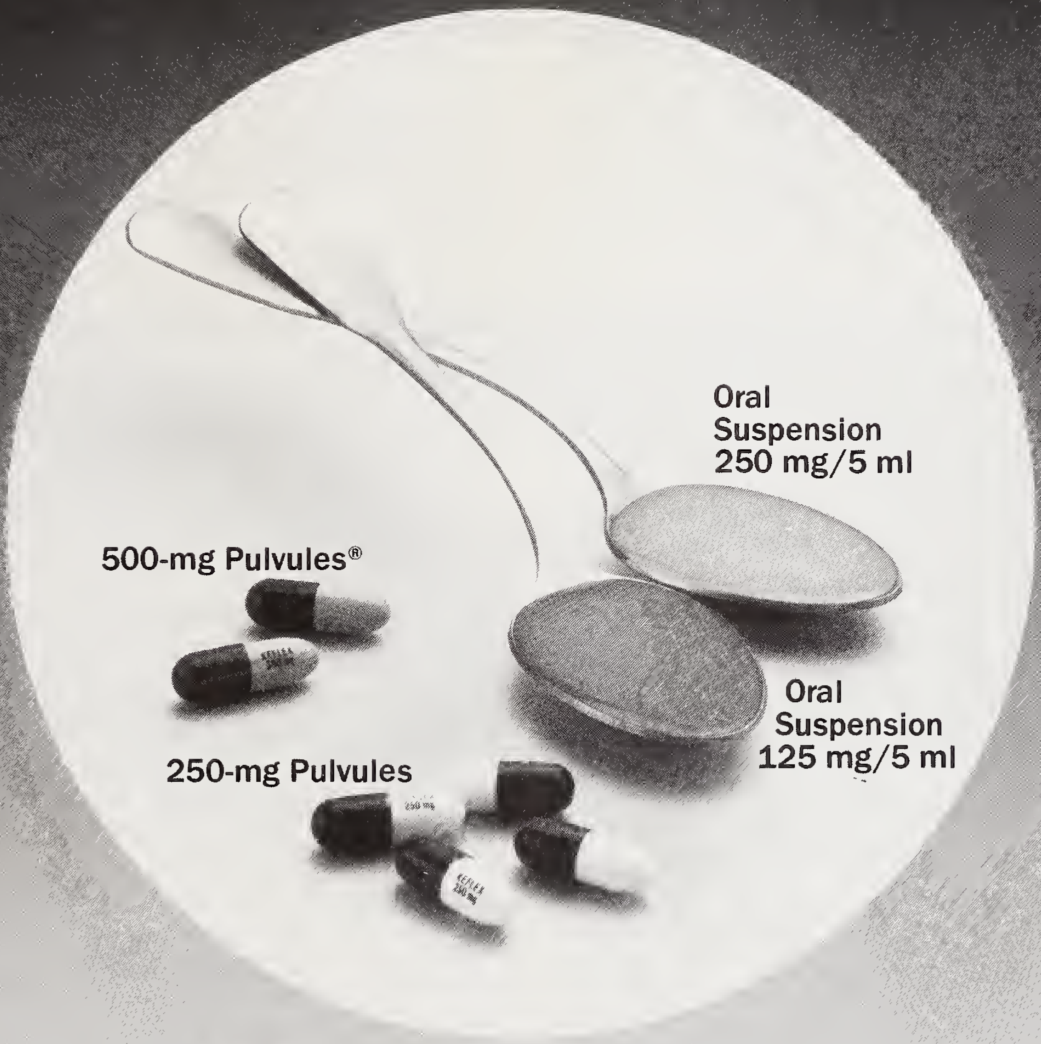

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CASE REPORT: TETANUS AS A COMPLICATION OF SMALL BOWEL OBSTRUCTION

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Our patient developed severe tetanus from a small bowel infarction without a penetrating wound. The patient had multiple risk factors associated with poor prognosis. We believe this is the first such patient to survive.

Once a major cause of wartime and trauma mortality, tetanus in the United States had diminished in importance to a deadly clinical curiosity. Although roughly half a million cases a year worldwide are reported,[1] many physicians in the United States never have seen a case because of the highly effective tetanus toxoid immunization. In the first 46 weeks of 1981, the *Morbidity and Mortality Weekly Report* reviewed only 55 cases.[2] Although most cases occur in nonimmunized or partially immunized individuals,[3] the disease may occur in fully immunized people. In a review of 2,449 cases over 68 years at the Charity Hospital in Louisiana, the incidence dropped from 3.5 cases/1,000 admissions in 1914 to 0.02 cases/1,000 admissions in 1974. Despite the decline, case mortality has remained greater than 50 percent.[4]

Most cases of tetanus are assumed to result from invasion of the organism through a contaminated wound, cut, or burn. There have been cases reported resulting from rubber band ligation of hemorrhoids,[5] and as a complication of ischemic gangrene of the toe of a nontraumatic nature.[6]

We report a most unusual presentation.

CASE REPORT

A 74-year-old white male was admitted with a 17-hour history of acute lower abdominal pain and vomiting. One year prior to admission, the patient underwent an exploratory laparotomy for a small bowel obstruction as a consequence of diverticulitis. The medical history was otherwise negative except for severe osteoarthritis of the left hip requiring 3.25 to 3.9 g of aspirin a day.

On physical examination, his temperature was 99.6°F with otherwise normal vital signs. The abdomen was mildly distended with bilateral lower quadrant guarding and hyperactive bowel sounds. Stool was negative for occult blood; hemoglobin was 15.1 gm/dl; WBC was 13, 6 with 70% polymorphonuclear neutrophilic leukocytes and 10% bands. SMA 18 and amylase were normal. Flat and upright x-rays of the abdomen revealed dilated loops of small bowel, but no free air was observed.

The patient was treated with intravenous fluids, cefoxitin, 1 gm every 6 hours, and nasogastric suction. The following day, the patient was afebrile and the abdomen was less distended. A gastrograffin enema revealed diverticulitis without perforation. Over the ensuing four days, the small bowel obstruction failed to resolve despite placement of a cantor tube. Exploratory laparotomy was scheduled for the fifth day.

The evening prior to surgery the patient experienced extreme respiratory difficulty, requiring ventilatory support. Closer observation revealed trismus and facial grimacing. Between episodes the patient was able to relate that he could not oppose the violent contractures of his jaw. He soon developed risus sardonicus and opisthotonos. Careful examination of the skin failed to reveal any puncture wounds. Blood cultures were negative. It was later verified that the patient had not received primary tetanus immunization.

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Therapy was initiated with 6,000 units of tetanus hyperimmune globulin, passive tetanus toxoid immunization, intravenous penicillin, and nasotracheal intubation in the operating room. An exploratory laparotomy performed at that time revealed a 6-inch segment of infarcted small bowel secondary to the volvulus approximately 18 inches proximal to the ileocecal valve. The bowel was reanastomosed primarily after resection of the segment with 3-inch margins on both sides of the infarction. Gram stain of the necrotic segment contained gram-positive rods. Postoperatively the patient's tonus could not be controlled with high-dose intravenous diazepam. A continuous pancuronium infusion was started to maintain complete paralysis. A morphine infusion assured amnesia, sedation, and analgesia.

This reports shows the need for questioning the immunization status of patients with a propensity toward anaerobic infection.

The patient remained on total support for nine weeks. The pancuronium was tapered every four days to assess his unparalyzed state. He sustained the sequelae of such support—atelectasis, pneumonia, fluid and electrolyte imbalance, inadequate nutrition, and antibiotic-related diarrhea. Paroxysmal disturbances in autonomic function common in tetanus were manifested in several ways. Wide swings in systolic blood pressure, often from 200 mm to 70 mm or as commonly from 70 mm to 200 mm were observed. These were treated with propranolol or fluids. Bursts of sinus tachycardia also responded to this therapy. Thermoregulatory dysfunction with abrupt swings in temperature to 104°F occurred early in the hospitalization and responded to active cooling.

The patient eventually was weaned from all support systems and underwent intensive rehabilitation. He was discharged on hospital day 92. He has returned to his law practice without neurological sequela. As intended, he has no recall of the intensive care stay.

DISCUSSION

Clostridium tetani has been isolated in human feces at rates up to 25 percent.[3] It has been noted that the presence of necrotic tissue may enhance the reversion of the spores to the vegetative form.[7] In the anaerobic environment of a segment of compromised small bowel, this reversion easily can be envisioned. The disease is caused by tetanospasmin, one of the exotoxins elaborated by the bacilli, *Clostridium tetani*. The toxin is spread hematogenously throughout the body, reaching the central nervous system and binding to the gangliosides of the synaptic membranes. By preventing release of the inhibitory neurotransmitter glycine, unopposed central nervous system stimulation of musculature ensues.[3] The toxin also acts directly on muscle to cause contraction. In addition, there is a marked but poorly understood effect on the sympathetic nervous system. Circulating toxin can be cleared from the blood by the administration of antitoxin (tetanus immune globulin), but this will not affect the toxin already bound to the synaptic membranes. Thus, in effect, the progression of the disease can be arrested, but resolution of the problem must come with the slow and complete dissociation of the toxin.

The diagnosis is a clinical one and the presentation is amazingly consistent. In a series of 100 patients with generalized tetanus, all but one had trismus.[8] The disease must be thought of whenever a patient presents with trismus and muscle spasm. Other causes which may mimic the disease, such as strychnine poisoning, phenothiazine reaction, meningitis, epilepsy, and hypocalcemic tetany must be ruled out.

The case raises a question of immunization of the elderly. Crossley et al.,[9] in a study of 183 urban adults, found protective levels of antibodies present in only 41 percent of the males over 60, and 29 percent of the females in the same age group. While some have advocated immunization updates on the geriatric population,[10,11] others have argued that this would not be cost effective.[12] We currently are studying the immunization status of patients in our area, and further will address this question.

There have been two previous reports of tetanus arising from unperforated infarcted bowel. Clay and Bolton described a surviving patient who developed a mild case of generalized tetanus four days after resection of gangrenous ileum.[13] Cultures of peritoneal fluid taken prior to manipulation of the bowel were positive for *Clostridium tetani*. Calvet reported a patient who developed trismus prior to bowel resection for a gangrenous terminal ileum.[14] This patient died shortly after the operation.

Our case is unique in a number of ways. To our knowledge this patient is the first survivor of tetanus arising from unperforated infarcted bowel in whom the symptoms were present prior to resection. The prognosis of tetanus is determined by a number of risk factors. Mortality increases with age, the severity of underlying illnesses, and the size of inoculum, and directly is related to the rapidity of onset of symptoms. An 80 percent mortality has been reported where the latter is less than 24 hours.[4] Our patient had all the aforementioned risk factors.

Although antibiotic therapy was begun promptly upon admission and several days prior to actual tetanus, it was not effective in preventing the disease. This case emphasizes the need for careful questioning regarding the immunization status of any patients with a propensity toward anaerobic infection. It also supports previous findings that recovery from severe cases is complete and without serious neurological sequelae.[15] Treatment should be initiated early in an intensive care unit. The combination infusion of pancuronium and morphine was most efficacious and titratable. Strict attention to detail is mandatory for a favorable outcome.

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OPINION: DRG—AN IMPETUS FOR CHANGE IN HEALTH CARE DELIVERY

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As a result of the DRG system, changes in health care delivery can be anticipated. Clinical research, new technologies, and medical education costs have been cited. We must anticipate that relationships between physician and hospital will change. Regionalization and promulgation of standards also may occur.

Evidence is accumulating that the federal DRG system will change health care delivery. Anderson and Steinberg have shown its potential impact on the introduction of new technology.[1] More recently, Yarbro and Mortenson have pointed out the deleterious effects of the system upon clinical research.[2] The current session of the U.S. Congress will debate medical education costs which have been highlighted by prospective payment.

More long-term effects can be anticipated. The system has not been operational long enough for any long-term evaluation. New Jersey's experiment is so different from the federal program that comparisons are not valid. Nonetheless, patterns may be emerging which can have a great impact on patient care. Where a hospital accrues losses due to stringent reimbursement, it will attempt to charge more to other patients or their insurance carriers. Evaluation of the true outcome of prospective DRG pricing must await universal adoption of the system, a trend which is beginning.

We already are witnessing a revolution in hospital administration. Hospital administrators are reassessing and enhancing their roles. Sophisticated hospital management personnel and advanced informational systems are evolving. Henceforth, administrators will use fiscal yardsticks to evaluate both physicians and services. The DRG system allocates a sum of money to a hospital based upon the patient's principal diagnosis. Before DRG, we had a system in which the hospital reimbursement was based upon reasonable expenditure per patient. Costs were studied only to recapture expenditure. In the old system, the cash flow was virtually limitless. Wants and needs were matched easily with dollars. The new system is quite stringent. Fiscal restraints will limit the fulfillment of requests. Since a hospital receives one set amount, expenditures must be examined and compared with those of many hospitals. It is anticipated that the system will tell which physician and service are creating a profit and which are not. A physician will be measured on his net income to the institution. Computers easily will analyze patient resource consumption and measure this against payment to the hospital. Based upon these figures, physicians will be categorized as "DRG profitable or unprofitable." Doctors whose profile shows them to be profitable will get the equipment which they request to keep them happy and in the fold. Those who are shown to be unprofitable can expect, at a minimum, an admonishing letter reminding them how much their patients are expending in contrast to reimbursement. Their requests for equipment and services quietly will be ignored and any threats that they will take their patients elsewhere will be viewed optimistically.

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*Dr. Rineberg is an orthopedic surgeon. Correspondence may be addressed to Dr. Rineberg, 303 George Street, New Brunswick, NJ 08901.

The informational systems will aid in the production of verifiable and precise patient data. This is required in the first instance to maximize reimbursement. Secondly, it will be essential for meaningful cost analysis. In addition, the systems will break down the patients' resource consumption to a finite degree and attempts will be made to eliminate waste. Both services and equipment will be viewed from the standpoint of profitability rather than enhanced image.

Careful cost analysis and reimbursement potential will precede major purchases. Items which can show overall savings by eliminating other expenses will have increased market potential. The world still will beat a path to buy a better and even more expensive mouse trap. But, as a tradeoff, the new device may have to use cheaper cheese.

PROSPECTIVE PAYMENT

Prospective payment inevitably will lead to increased regionalization. For years, some physicians and many economists have advocated regionalization as the most efficient method of delivering tertiary care. They believe that, though somewhat inconvenient, it would increase the quality while lowering the cost. With DRG, most institutions will not be capable of supporting services which rarely are used. With the prior system of pass-through of expenses, there was no incentive to discontinue or even to identify a service which was profitable. An unprofitable service always could survive with enough pressure either internal or external to the institution. Political forces will continue to impact only for the short term. The institution's future ability to survive will limit the frequency of continuing "loss leaders." Areas of activity which cannot be economically justified will be at risk. The prudent administrator will take measures to avoid the patient with a rare or difficult diagnosis which requires costly equipment. Hospitals no longer will be able to use expensive equipment sporadically and make it cost effective. Conditions which cannot be managed expeditiously and whose costs exceed the norm will be funneled to other institutions.

For example, the patient with multiple injuries no longer may be treated in the average hospital. These patients require long stays and special medical and nursing talent. Units with highly technical equipment and specialized staff will treat the patient more expeditiously. The equipment can only be efficiently used in volumes which justify cost. Also, long hospitalizations for which there is scant reimbursement will not be accepted readily. Physicians will be required to transfer patients to hospitals specializing in that particular type of care. Shared services will be used to reduce costs on expensive items in order to keep both physicians and patients in more remote areas. It no longer will be prudent for hospitals just a few miles apart to have their own high-priced laboratory, laundry, or even informational system. Though sharing will, at times, put strains upon the user and wear administrative patience quite thin, it does provide some answers to thorny problems.

STANDARDS OF CARE

Promulgation and acceptance of standards of care can be anticipated. There is little doubt that the DRG system calls for the creation of standards. Presently, a practitioner's approach to a problem can vary immensely. Witness that the average length of stay on the west coast is almost half that of some eastern states. Though some surgical specialties already have created and promulgated standards, others have been reluctant to do so. Standards are published primers which describe the care which should be rendered once a diagnosis has been determined.

COST OF TREATMENT

The DRG system allows a predetermined global price for a particular diagnosis, not a treatment. There will be pressures to employ only a certain modicum of care which, at the very least, will be influenced by price. With reimbursement based on diagnosis rather than procedure, the treatment will tend to be standardized. Physicians will have to expedite patient care by using certain predetermined and accredited tests or procedures. Other parameters of treatment will be contraindicated on the basis of cost, redundancy, or low yield. Despite their reluctance, specialty groups with their broad-based scientific community will be called upon to create these standards. The business community will be affected and definitely ambivalent when standards are introduced. Modalities of treatment no longer may be marketable due to lack of accreditation. On the other hand, other new technologies could burst forth if rapidly incorporated into the standards. The medical community also will look upon standards with ambivalence. The standards which are created inevitably will lead to uniformity of medical practice. Many legal issues will surface. Law suits may be instituted wherever deviation compromises results. Additional litigation can be anticipated to force the modification of existing standards. Possibly the most undesirable aspect of a printed standard is its ridigity and intolerance of deviation. In this manner, standards truly thwart innovation and progress. Standards soon will be commonplace.

HOSPITAL SURVIVAL

Lastly, we surely can anticipate that some hospitals will not survive the crunch. Innovators and advocates of the DRG system feel that the country is overbedded. (Please take note that it was the federal planners and Hill-Burton funds which got us into this condition in the first place). Some institutions will grow because of their specialized mission. However, since the financial pot is capped, others will wither and die. Institutions with weak administrators, feuding medical staffs, low census, or especially a combination of these, are extremely vulnerable. Once the beds are lost, it is inconceivable that they ever will be reinstated.

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3. Rineberg BA: DRG-New Jersey and the federal system. *J Med Soc NJ*:81:406-408, 1984.

CLINICAL NOTE: SQUIRRELS IN NEW JERSEY

LEON G. SMITH, M.D., NEWARK*

Squirrels, potentially dangerous animals, are overrunning New Jersey. The author describes the problem and possible solution.

Squirrels are overrunning New Jersey and pose a potential source of ectoparasitic problems. Tree squirrels including the gray, fox, red, and flying species are entering attics seeking new homes as we deforest the state. In these buildings they nest, gnaw on wood, and chew on electric wires and cables. They are noisy and if cornered can cause serious bites and literally destroy a household. More importantly, they carry the sylvatic plague, especially the black flying species, and relapsing fever. Antibodies to encephalitis virus Powassan and the virus of Colorado tick fever have been isolated from red squirrels.

To achieve control, one must search diligently for the small hole in which they enter the building. Nearby trees should be cut 10 feet from the building and a 2-foot wide metal band around the tree trunk, 3 feet off the ground, can keep the squirrel from climbing the tree. Once one squirrel enters the building, others will follow and nest together producing much destruction and possibly disease.

Once in the building, they are difficult to eradicate despite liberal uses of naphthalene and paradichlorobenzene. Ammonia or pentachlorophenol produces quicker results. All openings going to the building should have wire shields to repel this entry port.

When all of these measures fail, then trapping with rat traps bated with prunes and nuts placed in trees or fences can be effective. Poisons such as anticoagulants and tracking powders do not work well with squirrels. They hoard the bait rather than eat it.

Since squirrels are a protected species, killing them with traps or a gun requires a local permit. Trapping them live, using peanuts or peanut butter, may be more costly but more effective in the long run.

In conclusion, the squirrel is a potentially dangerous animal when allowed in the house and all measures must be used to eliminate them from causing local destruction and potentially lethal diseases.

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*Dr. Smith is Director of Medicine, Saint Michael's Medical Center. Correspondence may be addressed to Dr. Smith, Saint Michael's Medical Center, 268 Martin Luther King Jr. Blvd., Newark, NJ 07102.

DOCTORS' NOTEBOOK

Trustees' Minutes; Current Legislation; UMDNJ Notes; MSNJ Auxiliary; AMNJ Report; Physicians Seeking Location in New Jersey

Trustees' Minutes October 20, 1985

A regular meeting of the Board of Trustees was held on Sunday, October 20, 1985, at the Executive Offices in Lawrenceville. Detailed minutes are on file with the secretary of your county society. A summary of significant actions follows:

Report of the President . . .
(1) Commissioner's Retreat . . . Received a summary of the issues addressed at the Commissioner of Health's Retreat; issues included: hospital capital allocation, projected hospital bed excess, potential reimbursement shortfalls, recommended solutions for managing shrinkage of the hospital system, certificate of need legislation, uncompensated care, and graduate medical education.
(2) Essex County Medical Society Program on HMOs . . . Was informed of the possibility of an educational seminar on HMOs similar to the program provided by the Essex County Medical Society on October 10.

Report of Executive Director . . .
(1) MSNJ Membership . . .
(a) MSNJ Paid Membership . . . Noted that paid membership on September 30, 1985, was 7,464.
(b) Members Dropped for Nonpayment of Dues . . . Noted that membership recruitment should be the foremost priority during this current administrative year.
(c) Commission for Collecting MSNJ Dues . . . Noted an adjustment was made in commission amounts to be paid for 1986 dues.
(2) MSNJ Financial Statements . . . Noted that the financial statements for the period ending September 30, 1985, were approved.
(3) Certificate of Need . . .
(a) Legislation . . . Noted that S-2869 is out of committee and a Senate vote could be taken at any time.
(b) General Repeal . . . Awaiting the results of a questionnaire sent to administrators of New Jersey hospitals, hospital governing boards, and NJHA to ascertain whether or not they are in favor of the repeal of the certificate of need law.
(c) Meeting with Statewide Health Coordinating Council (SHCC) . . . Noted that another meeting was planned to discuss the certificate of need issue as well as the topic of HMO-PPO and the impact of these delivery systems on rate setting and planning programs.
(4) Subordinated Loan Certificates . . . Noted that the IRS is processing assessments in those instances where the statute of limitations has not expired for purchasers of subordinated loan certificates with deficiency notices.
(5) Contracting Checklist . . . Noted that a "Contracting Checklist" brochure has been sent to MSNJ membership.
(6) Professional Liability Rates . . . Noted that the Board of Governors of MIIENJ and the Board of Directors of New Jersey Medical Underwriters, Inc. have approved a rate filing which will effect a 27 percent increase in professional liability rates.
(7) Insurance Commissioner's Task Force on Malpractice . . . Approved MSNJ's response to the Commissioner of Insurance regarding the report of the Task Force.
(8) Alfone *v* Area VII PRO, New Jersey Blue Cross, Monmouth Medical Center, MSNJ et al. . . . Noted that MSNJ is included in this lawsuit because of its participation in the DRG second level appeal.
(9) Preadmission Certification Program . . . Noted that in an attempt to resolve the possibility that preadmission certification programs are not being managed through utilization review organizations, the Insurance Commissioner has scheduled a meeting where MSNJ and others will be represented.
(10) Medicare 45-Day Freeze Extension . . . Were provided with a communication from the AMA explaining the present status of physician reimbursement under the Medicare program including the 45-day freeze extension.
(11) Proposed Regulations . . .
(a) Physical Therapy Practice . . . Will forward MSNJ's position on the proposed rules to the State Board of Physical Therapy, the State Board of Medical Examiners, the Attorney General, Governor Kean, the President of the Senate, and the Speaker of the House.
Note: MSNJ's position is that physical therapy is appropriate after a licensed physician has examined the patient, made a diagnosis, and prescribed the therapy, either verbally or by written order.
(b) Delegation of Nursing Tasks . . . Directed that MSNJ's objection to the proposed regulation be forwarded to the State Board of Nursing, the State Board of Medical Examiners, the NJ Commissioner of Health, the Attorney General, and Governor Kean.
Note: The proposed regulation would establish that a registered professional nurse may assign the performance of certain nursing functions to other nurses and/or ancillary personnel.

New Jersey Hospital Association . . . Offered congratulations to Dr. Watson on his appointment to a two-year term on the Regional Advisory Board of the American Hospital Association.

AMA Delegation . . .
(1) Nomination of Candidates for Positions on AMA Delegation . . . Referred the following recommendation to the Committee on Long-Range Planning and Development and to the Committee on Revision of Constitution and Bylaws:

That the nominating procedure for AMA delegate positions be changed and that consideration be given to the development of a system whereby the Board of Trustees rather than the Nominating Committee would recommend nominations for these positions to the House of Delegates. (If adopted, a Bylaw change would be required.)

(2) **Delegation Officers** . . . Referred to the Committee on Long-Range Planning and Development and to the Committee on Revision of Constitution and Bylaws for discussion and report on the current procedures of electing the chairman and vice-chairman of the delegation.

(3) **Evaluation of Performance and Attendance/Role of Delegation** . . . Approved the following recommendation:

> That the Board of Trustees, with input from the delegation, develop guidelines for the function of the AMA delegation at AMA meetings and during the intervening time between AMA meetings.

(4) **Preparation for AMA Meetings** . . . Noted that the delegation should review the AMA handbook to develop strategy in an effort to be more effective at AMA meetings.

(5) **Unit Rule** . . . Approved the following recommendation:

> That the Board of Trustees approve the policy that the unit rule shall apply when two-thirds of the delegation vote to support a given candidate for AMA office.

(6) **Caucus Groups** . . . Approved the following recommendation:

> That the Board of Trustees empower the president of the Society, the chairman of the delegation, and the Executive Director, to investigate the possibility of joining one of the existing coalitions.

Committee on Impaired Physicians . . . Approved the following recommendation:

> That the brochure, "Guidelines for Hospital-Based Wellness Committees," be approved by the Board of Trustees.

Special Committee on Emergency Medical Care . . . Approved the following recommendation:

> That MSNJ adopt the guidelines developed by the American College of Emergency Physicians for freestanding emergency centers.

Audit Review Committee . . . Approved the following recommendation:

> That the audited financial statements be accepted.

Old Business . . . Agreed to accept the revised conflict-of-interest disclosure form recommended by Dr. Watson.

Current Legislation

The Board of Trustees of the Medical Society of New Jersey approved the recommended positions on the following bills of medical interest:

S-3055-Pallone—Anatomical Gifts. Provides that anatomical gift cards used by prospective donors shall be mailed to the State Police and recorded by them. The bill is intended to expedite organ donations. **Approved.**

S-3064-Zane—Financial Assistance for Organ Transplants. Creates a Commission to accept applications from teaching or affiliated hospitals to perform transplants. Based on the patient's financial need, a grant of up to $200,000 may be authorized. The Commission would consist of the Commissioner of Health, two deans of New Jersey Medical Schools, and two licensed physicians knowledgeable in transplant procedures. **Disapproved,** as written. The Council approves the intent of the legislation, but the practicality of the bill is not clear. The Council recommends that the Board of Trustees develop a policy on this topic with referral to existing Committees for report and recommendation.

S-3178-Connors—Hazardous Substances in Workplace. Requires the Department of Health and the Department of Environmental Protection to develop a single workplace survey to be sent to employers. **No Action.**

S-3203-O'Connor—Board of Chiropractic Examiners (A-3593). Creates a separate Board of Chiropractic Examiners within the Division of Consumer Affairs. **Active Opposition,** because there is no demonstrative need for this legislation. Chiropractors are supervised adequately under the auspices of the New Jersey State Board of Medical Examiners.

S-3212-Codey—Mental Health Coverage—Blue Cross. Requires hospital service corporation contracts to provide a minimum of 60 days inpatient care or 120 days of partial care during a 12-month benefit period. **Approved.**

S-3213-Codey—Mental Health Coverage—Blue Shield. Same as **S-3212** except it applies to medical service corporations. **Approved.**

S-3214-Codey—Mental Health Coverage—Group Contracts. Same as **S-3212** except it applies to group contracts. **Approved.**

S-3215-Codey—Mental Health Coverage—Commercial Carriers. Same as **S-3212** except it applies to commercial carriers. **Approved.**

S-3217-Codey—Mental Health Coverage—HMO. Same as **S-3212** except it applies to HMOs. **Approved.**

S-3256-Costa—Uniform Anatomical Gifts. Authorizes the medical examiner to remove eye tissue if the deceased had made a gift of his/her eyes, provided the procedure does not interfere with the course of any subsequent investigation or alter the decedent's postmortem facial appearance. **No Action.**

SJR-65-Cowan—Long-Term Care. Creates a commission to study the feasibility of mandating group health plan coverage of long-term care. **No Action.**

S-3302-Dumont—Immunity for Acts of Environmental Contamination. Exempts public entities and public employees from liability for injuries caused by the negligent contamination of the environment. **Active Opposition,** because the entire problem needs to be studied and resolved. Exemptions should not be granted to specific public entities and employees.

S-3313-Costs—Senior Citizen Respite Program. Requires the Commissioner of Health to contract with a general hospital for a pilot program to dedicate up to 20 of its unused beds to provide day and weekend care for the elderly. No certificate of need would be

ARE YOU MOVING?

If so, please send a change of address to *NEW JERSEY MEDICINE,* Medical Society of New Jersey, Two Princess Road, Lawrenceville, NJ 08648, at least six weeks before you move.

Name ____________________

Address ____________________

City ____________ State ______ Zip ______ required and rates would be under the review of the Rate Setting Commission. **Action Deferred,** pending further information from MSNJ's Council on Medical Services.

S-3368-Graves—Drug Abuse. Amends existing law to prohibit the manufacture and distribution of drugs designed to produce an effect similar to that of a controlled substance unless the activity is exempt under federal law. **Approved.**

A-2192-Penn—Motor Vehicle Vision Examination. Provides that a licensed ophthalmic dispenser may certify successful completion of the required visual examination for an operator's license. **Disapproved,** current law is preferable at this time. Motorists may feel they are receiving a quality eye examination when, in fact, the examination merely consists of an eye chart reading test and a test for peripheral vision and depth perception.

A-2554-Doria—Professional Boards. Creates a Review Commission within the Division of Professional Boards to study and comment on various licensing proposals. **Approved.**

A-2557-Zimmer—Emergency Services. Creates an intermediate EMT unit and type of personnel (companion to A-2005 of 1984). **Approved,** provided physician supervision is stressed.

A-2872-Felice—Methadone Registry. Establishes a central registry within the Department of Health of patients enrolled in methadone maintenance programs. **Disapproved,** because this legislation would be impractical and expensive—also such a registry reflects an approach to a problem that does not exist.

A-3543-Kalik—Dispensing of Prescription Drugs. Intends to alleviate the inability of certain elderly patients to open their prescription containers that are closed with childproof cap. Amendment to the bill would require a new box and statement on prescriptions. **No Action.**

A-3593-Zangari—Board of Chiropractic Examiners (S-3203). Creates a separate Board of Chiropractic Examiners within the Division of Consumer Affairs. **Active Opposition,** because there is no demonstrative need for this legislation. Chiropractors are supervised adequately under the auspices of the New Jersey State Board of Medical Examiners.

A-3684-Adubato—Sulfites. Prohibits wholesale or retail distributors of food or food products from adding sulfites to their product. **No Action.** Dr. Allen voted in the negative and asked to be so recorded.

A-3700-Muziani—Medical Advice. Requires physicians to advise pregnant patients of the effects of alcohol and certain drugs on a fetus. The woman shall sign a form confirming the advice was given. The physician is to retain the original and provide the patient with a copy. **Active Opposition,** because this legislation interferes with the regulatory authority of the State Board of Medical Examiners and is beyond the scope of sound legislative policy.

S-3715-Kline—Water Storage Tanks. Provides that the Department of Environmental Protection shall inspect and certify the condition of water in tanks exceeding 5,000 gallons. **No Action.**

A-3723-Felice—Childbirth. Requires hospitals to allow fathers to observe deliveries as long as the mother and the attending physician or midwife agree. **Disapproved,** because hospitals and their medical staffs should be allowed to set their own policies.

A-3737-Doria—Professional Review. Grants immunity to members of ethics, grievance, judicial, or professional relations committees, a technical amendment. **Approved.**

A-3748-Vainieri—Blood Banks. Requires blood banks to screen all blood for antibodies to human T-cell lymphotropic virus, type III (HLTV-III). **No Action.**

A-3776-Muhler—Deceptive Practice. Provides that anyone who sells drugs, devices, or provides treatments knowing they are unsafe, ineffective, or unproved for safety or efficacy is guilty of a crime in the fourth degree. **Action Deferred,** pending further information from the sponsor of this bill as to its clarification and context. The bill, as written, is too vague and too general.

A-3798-Otlowski—Medical Service Corporation/Drug Abuse Treatment. Requires all group and individual medical service corporations to cover the expenses of treatment for drug abuse as for any other illness. **Approved.**

A-3799-Otlowski—Drug Abuse Treatment/Commercial Carriers. Same as **A-3798,** but applies to commercial carriers. **Approved.**

A-3800-Otlowski—Group Health/Drug Abuse Treatment. Same as **A-3798,** but applies to group coverage. **Approved.**

A-3801-Otlowski—HMO Coverage/Drug Abuse Treatment. Same as **A-3798,** but applies to HMOs. **Approved.**

A-3802-Otlowski—Group Health Insurance/Drug Abuse Treatment. Requires Blue Cross to cover the expense of treatment for drug abuse as for any other illness. **Approved.**

A-3824-Franks—Regulatory Efficiency. Creates a study commission to review the regulatory process. The commission is to determine whether the regulatory functions of state agencies are necessary, straightforward, nonduplicative, of minimal expense to the regulated entity, and adequate to protect the public interest. **Active Support.**

A-3889-Perun—Kawasaki Disease. Requires the Department of Health to institute screening and education programs to promote the early detection and treatment of Kawasaki disease. **No Action,** advises sponsor that a disease study and education program of Kawasaki disease should be conducted through UMDNJ not through legislative process.

A-3893-Randall—Emergency Medical Services. Provides for the establishment of at least two hospital-based EMS helicopter response units. **Approved.**

A-3902-Muziani—Tanning Lights. Permits the use of ultra-violet tanning lights in licensed beauty culture shops. **Disapproved,** because the use of ultraviolet tanning lights is potentially dangerous.

A-3962-Marsella—Radiation Protection. Adds medical physics and epidemiology to the types of training of members of the Radiation Protection Commission. Also adds two members to the Commission. **Approved.**

A-3998-Bryant—DRG Exemption. Designates Cooper Hospital as the "South Jersey" specialty acute care children's hospital and requires the Department of Health to develop adequate reimbursement rates. **No Action,** letter to Governor and sponsor of bill, re obvious defect in DRG system.

A-4000-Baer—Hazardous Substances. Creates a plan to list the extraordinarily hazardous substances and to develop an assessment of catastrophic risk to the public health and a design for risk prevention and management. **Approved.**

A-4006-Perun—AIDS. Establishes an AIDS Task Force to compile and analyze data to advise the state regarding prevention, control, and public education. **Conditional Approval,** pending amendment of the bill to include provisions for epidemiological followup on all AIDS cases.

A-4007-Perun—AIDS. Appropriates an additional $5 million to UMDNJ for the AIDS resource center. **Conditional Approval,** pending amendment of the bill to include provisions for epidemiological followup on all AIDS cases.

A-4016-Pelly—Impaired Health Professionals. Establishes a 15-member Impaired Health Professionals Study Commission. MSNJ is to designate 1 of the members. **Active Support.**

A-4017-Frelinghuysen—AIDS. Appropriates $1.6 million for services to AIDS patients and their families. **Conditional Approval,** pending amendment of the bill to include provisions for epidemiological followup on all AIDS cases.

A-4032-Paterniti—CPR for Senior Citizens. Requires the Department of Health to establish a statewide program to train senior citizens in CPR. **No Action.**

A-4074-Paterniti—Pharmaceutical Information. Requires the Department of Health to provide statewide pharmaceutical information services for physicians and pharmacists. **Disapproved,** the Department does not have the resources and manpower to effect this function which is more appropriate for UMDNJ.

A-4119-Doria—Chiropractic. Creates a licensing board for chiropractic. Transfers all functions and responsibilities of the State Board of Medical Examiners regarding chiropractic to the new board. Removes the place for a chiropractor from the medical board. **Active Opposition,** because there is no demonstrative need for this legislation. Chiropractors are adequately supervised under the auspices of the New Jersey State Board of Medical Examiners.

A-4122-Ogden—Certificate of Need/Cardiac Surgery. Requires the Commissioner of Health to issue a certificate for cardiac surgery to hospitals that demonstrate that they have lawfully operated a catheterization facility which performed at least 750 procedures in the year following the year the Commissioner denied their application. **No Action,** MSNJ is opposed to the certificate of need.

A-4124—Ancora Developmental Center. Referred to the Council on Mental Health for an opinion. **Action Deferred,** pending further information from the Council on Mental Health.

A-4148, 4149, 4150, and 4151-Paterniti—Hospice Care. Requires insurers and HMOs to cover hospice care as defined by the Department of Insurance to include at least 210 days of care. **Approved.**

AR-146-Shusted—DRG for Physicians. Requests Congress to consider the recommendations of the American Academy of Family Physicians in establishing a federal physician DRG program. **Active Opposition,** the DRG program has conceptual design and operating flaws. It has not achieved the goals of cost effectiveness and quality assurance. It should be repealed, not expanded to physicians. It is impractical to impose on physicians.

AR-147-Shusted—DRG for Physicians. Requests the Assembly to consider the recommendations of the New Jersey Academy of Family Physicians before implementing a physician DRG program in New Jersey. **Active Opposition,** same as **AR-146.**

AR-157-Otlowski—Mental Health. Declares the opinion of the Assembly that adequate care should be provided to the chronically mentally ill residents of this state. **Approved.**

UMDNJ Notes

Stanley S. Bergen, Jr., M.D.
President

New Jersey's goal is to maintain its lead and progressive attitude in economic, social, and scientific development. More than a year ago, voters acted to do just that in the area of high technology by approving the $90 million jobs, science, and technology bond issue.

Since then, major strides have been made with respect to all of the bond issue's components, which were developed specifically to coincide with the state's existing industrial and academic strengths or to the technological fields envisioned to have the greatest potential for economic expansion. Of all the initiatives set forth, UMDNJ has a special role in the New Jersey Center for Advanced Biotechnology and Medicine (CABM) because of its potential impact on scientific research and health care in New Jersey.

Although construction of the Center's 50,000-square-foot core facility is not scheduled to begin for another year or so, the CABM took a major step forward a few weeks ago with the appointment of one of New Jersey's most eminent scientists to serve as its director. Aaron J. Shatkin, Ph.D., head of the department of Cell Biology with the Roche Institute of Molecular Biology in Nutley, is expected to fully assume his duties as director by early next summer, but already is working with the Center and its Scientific Advisory Board on design and governance issues, and research priorities.

The CABM will be administered jointly by UMDNJ and Rutgers, the State University, and will be built on a section of the Piscataway-based Busch campus shared by the two institutions. Under Dr. Shatkin's direction, the Center will complement the existing resources of the UMDNJ-Rutgers Medical School and Rutgers University's Waksman Institute of Microbiology—in addition to recruiting a staff of core scientists working in the area of advanced biology.

As physicians, we know the importance of keeping pace with the ever-changing health care field. In many respects, the CABM will enable us to set the pace. Together with scientists and technicians from other academic institutions and private industry, these researchers will attempt to accelerate basic research into clinically relevant and other applications. Dr. Shatkin's other responsibilities will include establishing and administering educational programs for undergraduate and graduate students.

The Center will be comprised of a $28.2 million core facility to be constructed between the medical school and the Waksman Institute; a $3 million satellite facility at Waksman for the production and separation of cells and cell products; and a $3.8 million clinical research center at Middlesex General-University Hospital, New Brunswick, the core teaching hospital of the UMDNJ-Rutgers Medical School.

MSNJ Auxiliary

Bernardine Moloney
President

The Auxiliary is planning an active and productive 1986. All are invited to join us for our annual legislative trip to Washington, D.C., scheduled for April 9-10, 1986; reservations are available on a first-come basis by calling the Auxiliary office.

Rather than visit our own legislators, many of whom have seen us before, we are attempting to make appointments with members of Congress who serve on key committees. Medicine's voice will be heard!

We will have dinner with, and be briefed by, a lobbyist from the AMA. Breakfast will be shared with a legislator who also will speak to us. Plan now to be a part of this interesting trip.

Additionally, all members are encouraged to attend the regularly scheduled Board meetings. November's meeting was followed by a program on mammography and self-breast examination; and our meetings in January and March also will provide some type of educational forum.

I wish everyone a happy and healthy 1986.

AMNJ Report

Dennis R. Filippone, M.D.
President

The Academy is delighted to announce that The Accreditation Council for Continuing Medical Education (ACCME) has approved our reaccreditation for a six-year period. This approval is the result of an extensive review by the ACCME's Accreditation Review Committee and authorizes the Academy to continue to sponsor accredited CME programs for physicians. The Academy originally was approved in 1972 and will be due for resurvey in late 1991.

For the second consecutive year, the Academy is the recipient of a grant from the New Jersey State Department of Health to provide an AIDS educational program to health professionals throughout the state of New Jersey. Hospitals and other health care institutions have been informed of the program and are presently signing up for lectures. We will be administering the lectures through our Roving Symposia series and expect about 30 programs.

The grant also includes the opportunity to sponsor a one-day major symposium on the subject.

Plans are underway to present a program on "AIDS and the IV Drug Abuser" on Wednesday, April 16, 1986, at our headquarters building in Lawrenceville. Dr. Daniel Greenfield will be program chairman and the meeting will be cosponsored by the Medical Society of New Jersey. In addition to plenary topics, there will be a series of workshops addressing counseling, school, ethical, economic, and screening issues.

On September 1, 1985, the Academy added the New Jersey Medical Directors Association to the growing list of societies receiving executive headquarter services from the Academy. On October 24, the Association held a successful meeting at our building on "Implementing Life-Sustaining Treatment Decisions in the Long-Term Care Facility" with over 160 in attendance. Dr. Morris Green is the current president.

The topic for the Academy's First Wednesday program for December was "The Corporate Evolution of Hospitals Through Multi-Hospital Mergers." Our guest speaker was Dr. Joseph Lindner who is the former chief executive officer of the Trimark Corporation. Dr. Lindner addressed a topic of major current interest to New Jersey physicians and hospitals.

Physicians Seeking Location in New Jersey

The following physicians have written to the Executive Offices of MSNJ seeking information on possible opportunities for practice in New Jersey. The information listed below has been supplied by the physician. If you are interested in any further information concerning these physicians, we suggest you make inquiries directly to them.

ANESTHESIOLOGY—John Junior, M.D., 9315 Ridge Blvd., Brooklyn, NY 11209. Wroclaw (Poland) 1980. Also, internal medicine. Board certified. Group, partnership, solo. Available February 1986.

S.H. Emami Kelishadi, M.D., 4201 Cathedral Ave., NW 222E, Washington, DC 20016. Tehran (Iran) 1957. Also, emergency medicine. Group or partnership. Available.

EMERGENCY MEDICINE—S.H. Emami Kelishadi, M.D., 4201 Cathedral Ave., NW 222E, Washington, DC 20016. Tehran (Iran) 1957. Also, anesthesiology. Group or partnership. Available.

Julian A. Sellek, M.D., 1501 South Greenway, Coral Gables, FL 33134. Salamanca (Spain) 1963. Also, family practice. Board eligible. Board certified (FP). Group, solo, partnership. Available.

FAMILY PRACTICE—C.C. Enu, M.D., c/o Dr. Foins, 291 Decatur St., Brooklyn, NY 11233. Albany Medical 1969. Board certified. Group, hospital, partnership, HMO. Available.

Lawrence S. Gordon, M.D., 1016 Spinnaker Way, Apt. G-1, Melbourne, FL 32935. Rutgers 1982. Board certified. Group, partnership, solo. Available spring 1986.

Debra T. Miller, M.D., 1428 D Mews Dr., Kansas City, MO 64131. Hahnemann Medical School 1982. Board eligible. Group or partnership. Available.

Julian A. Sellek, M.D., 1501 South Greenway, Coral Gables, FL 33134. Salamanca (Spain) 1963. Also, emergency medicine. Board certified. Board eligible (EMER). Group, solo, partnership. Available.

Susan F. Stickels, M.D., 1913 E. Kenmore Pl., Shorewood, WI 53211. Board certified. Group or HMO. Available.

Lew Herbert Valliant, M.D., 115 Mountain View Dr., Elkins, WV 26241.

THE MEDICAL SOCIETY OF NEW JERSEY
presents
Two Monthly TV Programs
PRESIDENT'S FORUM
Hosted by Ralph J. Fioretti, M.D.
President, MSNJ
STATE OF THE ART
Hosted by Frank Y. Watson, M.D.
Past-President, MSNJ

on the second and fourth Thursdays of each month at 1:00 p.m.

Topics will include:
"American Association of Retired Persons; An Impact on Medicine"; "The Society's Stand on Proposed Legislation"; "The PROs"; "Malpractice Tort Reform"; "Hospital Staff and Future of Medicine"; "Is There a Need for CNs?"

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Subscriber Hospitals

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Programs may be taped for alternate viewing times.

Queen's University (Canada) 1976. Board certified. Solo or group. Available.

INTERNAL MEDICINE—Arkady S. Fishman, M.D., 55 Nagle Ave., Apt. 1E, New York, NY 10040. Aktyubinsk Medical Institute 1969. Board certified. House staff, group, nursing home, industry. Available.

Irving S. Gottfried, M.D., M.P.H., 5700 Centre Ave., #1014, Pittsburgh, PA 15206. St. George's University (Grenada) 1981. Board eligible. Group, partnership, solo. Available July 1986.

John Junior, M.D., 9315 Ridge Blvd., Brooklyn, NY 11209. Wroclaw (Poland) 1980. Also, anesthesiology. Board eligible. Board certified (ANES). Group, partnership, solo. Available February 1986.

Anil Kapoor, M.D., 332 Lincoln Dr., Haworth, NJ 07641. All India Medical Institute 1966. Board eligible. Solo, partnership, group. Available.

Mark S. Noveck, M.D., 6807 Bonnie Ridge Dr., Apt. T-2, Baltimore, MD 21209. New York Medical College 1980. Board eligible. Group or partnership. Available.

Kevin W. O'Hara, M.D., 2250 Mt. Carmel Ave., Apt. A-2, Glenside, PA 19038. UMDNJ-New Jersey Medical School 1982. Board eligible. Group or partnership. Available May 1986.

Alka J. Patel, M.D., 1 John Och's Ct., Saddle Brook, NJ 07662. Topiwala National Medical College (India) 1983. Board eligible. Group or partnership. Available July 1986.

OPHTHALMOLOGY—Kenneth R. Brein, M.D., 55 Beacon St., Pittsburgh, PA 15217. University of Pennsylvania 1982. Board eligible. Group, partnership, solo. Available July 1986.

Andrew R. Gewirtz, M.D., 64 Pond Rd., Apt. G3, Woodbury, NY 11797. Chicago 1978. Board eligible. Board certified (PED) Single or multispecialty group. Available.

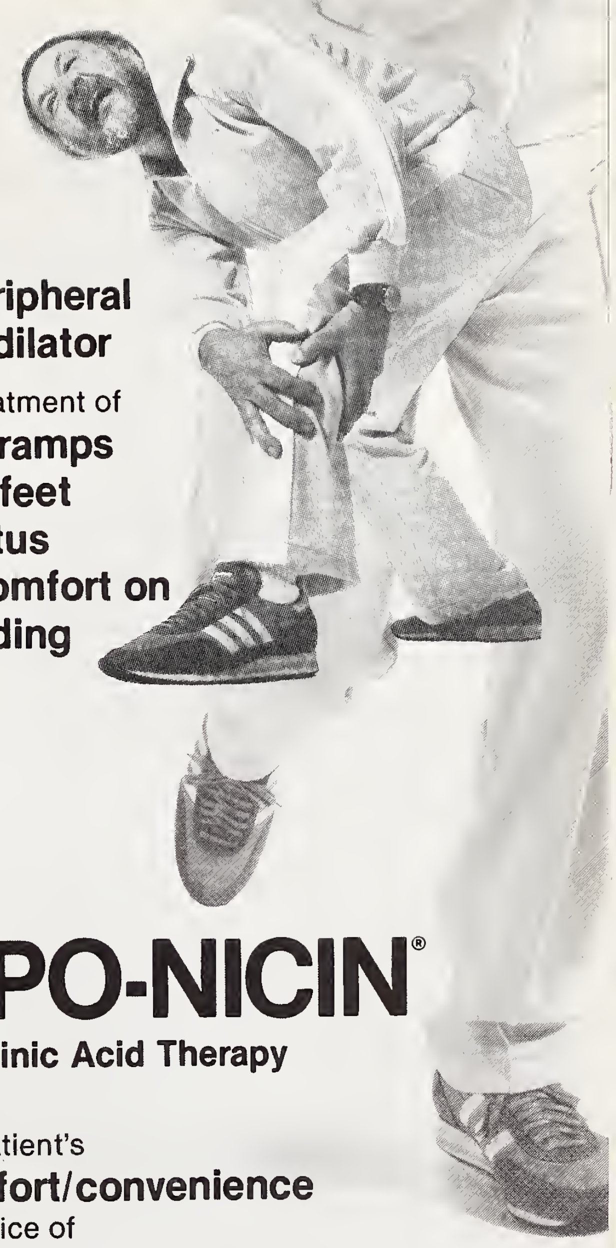
SURGERY—Derrick John Martin, M.D., 13-2 Granada Cres., White Plains, NY 10603. West Indies 1977. Board certified. Group, partnership, solo. Available.

Niranjan V. Rao, M.D., 3311 Clarendon Rd., Cleveland Heights, OH 44118. M.R. Medical College (India) 1976. Board certified. Also, vascular surgery. Group, partnership, solo. Available.

UROLOGY—Louis C. Galdieri, M.D., 1563 Hampton Knoll Dr., Akron, OH 44313. Guadalajara (Mexico) 1979. Group or partnership. Available August 1986.

John R. Scheuch, M.D., 116 Sycamore Rd., Jersey City, NJ 07305. St. George's University (Grenada) 1981. Board eligible. Partnership or group. Available July 1986.

Edward C. White, M.D., 5 Starlight Ct., North Babylon, NY 11704. Guadalajara (Mexico) 1979. Board eligible. Solo, partnership, group. Available.



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Nicotinic Acid	250 mg.
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Riboflavin (B-2)	2 mg.
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CARDIOLOGY UPDATE. . .

IS DESIGNED FOR THE PHYSICIAN AND PROVIDES AN INTENSIVE SURVEY OF THE CURRENT STATUS OF CLINICAL CARDIOLOGY. . .

WEDNESDAY, FEBRUARY 5, 1986

NUCLEAR IMAGING: ITS PLACE IN CARDIOVASCULAR MANAGEMENT TODAY

MODERATOR: ABDULMASSIH S. ISKANDRIAN, M.D.

3:00	CASE PRESENTATION: ACUTE MYOCARDIAL INFARCTION	*William S. Haaz, M.D.*
3:30	DISCUSSION, INCLUDING TECHNICIAL, THALLIUM & FIRST-PASS STUDIES	*Jonathan C. Felsher, M.D.*
4:00	CASE PRESENTATION: CORONARY BYPASS SURGERY	*John S. Owens, M.D.*
4:30	DISCUSSION, INCLUDING EXERCISE IMAGING	*Abdulmassih S. Iskandrian, M.D.*
5:00	QUESTIONS AND ANSWERS: THE AUDIENCE	

LECTURE HALL "A"—2nd floor New College Building, Hahnemann University
15th and Vine Streets, Philadelphia, PA

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• CME CATEGORY I CREDITS CERTIFIED •
WINE & CHEESE SERVED FOLLOWING CONFERENCE

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Next seminar Wednesday, January 22, 1986. Seminars monthly January through November except July and August. Registration fee of $95.00 includes course materials and approximately two hours hands-on computer time.

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CME CALENDAR

The following is a list of continuing medical education courses for the next two months. Contact the sponsoring organization for further information.

This list is compiled through the cooperation of the Committee on Medical Education of the Medical Society of New Jersey, The Academy of Medicine of New Jersey, the New Jersey Chapter of the American Academy of Family Physicians, and the Office of Continuing Medical Education of the UMDNJ. For information on accreditation, please contact the sponsoring organization(s), indicated by italics—last line of each item.

ANESTHESIOLOGY

February

8 **Clinical Anesthesia '86**
8-4 P.M.—NJ Medical School, Newark
(UMDNJ)

CARDIOLOGY

February

20 **Modern Outpatient Management of Congestive Heart Failure**
5 P.M.—Somerset Medical Center, Somerville
(Somerset Medical Center and AMNJ)

MEDICINE

February

Mini-Residency in Proctology and Diseases of the Colon
One Week Commitment—contact Overlook Hospital, Summit
(Overlook Hospital and AMNJ)

5 **Use of Exercise in Clinical Practice**
10:30 A.M.-12 noon—St. Mary's Hospital, Passaic
(AMNJ)

5 **Immunology Update**
1:30-2:30 P.M.—Essex County Hospital Center, Cedar Grove
(AMNJ)

5 **Scientific Meeting**
1:30-5 P.M.—Chilton Memorial Hospital, Pompton Plains
(NJ Society for Gastrointestinal Endoscopy and AMNJ)

5 **Internal Medicine Review Course**
12 4-7 P.M.—Middlesex General
Hospital, New Brunswick
(Middlesex General-University Hospital and AMNJ)

5 **Breast Cancer**
12 **Hodgkin's Disease and Other Lymphomas**
19 **Platelet Disorders**
26 **Lung Neoplasia**
8:30-10:30 A.M.—Alexian Brothers Hospital, Elizabeth
(Alexian Brothers Hospital and AMNJ)

5 **Medical Lecture Series**
12 10:30 A.M.-12 noon—Christ
19 Hospital, Jersey City
26 *(Christ Hospital and AMNJ)*

5 **Adolescent Medicine**
12 6-8 P.M.—NJ Medical School,
19 Piscataway
26 *(UMDNJ)*

5 **Adolescent Medicine**
12 6-8 P.M.—Halloran Plaza, Rt. 130
19 North, Pennsauken
26 *(UMDNJ)*

6 **Human Genetics**
13 4-6 P.M.—Institute for Medical
20 Research, Copewood St., Camden
27 *(Institute for Medical Research and AMNJ)*

8 **Research/Management—Alzheimer's Disease**
22 **Alternatives in Treatment of Abdominal Aortic Aneurysm**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

11 **Dermatology**
8-10 P.M.—Schering Corp., Kenilworth
(NJ Dermatological Society and AMNJ)

19 **Dermatological Conference**
6-9 P.M.—Rutgers Community Health Plan, 57 U.S. Hwy. #1, New Brunswick
(UMDNJ and AMNJ)

March

Mini-Residency in Proctology and Diseases of the Colon
One Week Commitment—contact Overlook Hospital, Summit
(Overlook Hospital and AMNJ)

5 **Adolescent Medicine**
6-8 P.M.—NJ Medical School, Newark
(UMDNJ)

5 **Adolescent Medicine**
6-8 P.M.—NJ Medical School, Piscataway
(UMDNJ)

5 **Adolescent Medicine**
6-8 P.M.—Halloran Plaza, Rt. 130 North, Pennsauken
(UMDNJ)

5 **Medical Lecture Series**
12 10:30 A.M.-12 noon—Christ
19 Hospital, Jersey City
26 *(Christ Hospital and AMNJ)*

6 **Human Genetics**
13 4-6 P.M.—Institute for Medical
20 Research, Copewood St., Camden
(Institute for Medical Research and AMNJ)

19 **Dermatological Conference**
6-9 P.M.—Rutgers Community Health Plan, 57 U.S. Hwy. 1, New Brunswick
(UMDNJ and AMNJ)

20 **Prostaglandins in Gastrointestinal Disease**
11 A.M.—St. Joseph's Hospital and Medical Center, Paterson
(St. Joseph's Hospital and Medical Center and AMNJ)

20 **Occupational Reproductive Hazards**
5-6:30 P.M.—Somerset Medical Center, Somerville
(Somerset Medical Center and AMNJ)

26 **I.A.B.P. and Medicine in China**
8-10 P.M.—Englewood Club, 115 East Palisade Avenue, Englewood
(Englewood Surgical Society and AMNJ)

NEUROLOGY/PSYCHIATRY

February

3 **Long-term Consequences of Sexual Abuse for a Boy**
8:15-10:15 P.M.—5 Fairfield Road, Montclair
(Essex Psychiatric Seminar and AMNJ)

6 **Case Seminars**
20 8-10 P.M.—312 Harding Drive,
South Orange
(Advanced Psychiatric Study Group and AMNJ)

March

3 **Medication of a Borderline Psychiatric Patient**
8:15-10 P.M.—9 Marquette Road, Upper Montclair
(Essex Psychiatric Seminar and AMNJ)

6 **Case Seminars**
20 8-10 P.M.—312 Harding Drive,
South Orange
(Advanced Psychiatric Study Group and AMNJ)

OBSTETRICS/GYNECOLOGY

March

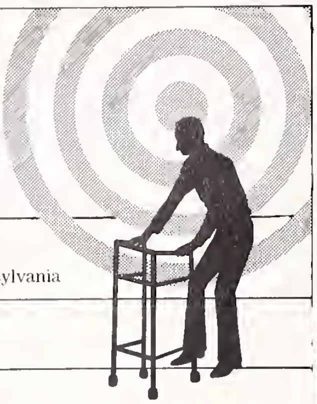
21 **Semmelweis-Waters Ob/Gyn**
22 **Conference**
23 Resorts International Hotel,
Atlantic City
(UMDNJ-NJ Medical School)

PEDIATRICS

February

4 **Pediatric Grand Rounds**
8:30-9:30 A.M.—Newark Beth Israel Medical Center
(Newark Beth Israel Medical Center and AMNJ)

6 **Management of Neonatal Problems**



COMMON CLINICAL CHALLENGES IN THE ELDERLY

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APRIL 3-5, 1986

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Common Clinical Challenges in the Elderly is designed to provide up-to-date information for the clinician who cares for the elderly in either ambulatory or inpatient settings. It is recommended specifically for internists, family practitioners and general practitioners.

Topics will include: GI disorders, cardiovascular diseases, infections, musculoskeletal disease, endocrine disorders, altered mental status, sleep disorders, foot problems, practical applications of exercise physiology, and drug prescribing.

Participants will receive 18-1/3 hours of Category I credit in Continuing Medical Education. (Reviewed and acceptable for 18 prescribed hours by the AAFP.)

For more information contact:

Ms. Nancy Spaw
Department of Medicine
Philadelphia Geriatric Center
5301 Old York Road
Philadelphia, PA 19141
(215) 456-2943

Philadelphia Geriatric Center

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in cooperation with

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(19 Successive Wednesdays)

4:00 P.M.-7:00 P.M.

at

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The course is designed to provide a comprehensive review of contemporary concepts in Internal Medicine. It is designed for practicing internists and family practitioners. The review will also be useful for residents. Syllabus material will be distributed during the course which will, when compiled, provide an extremely useful reference.

Course Chairman: Robert Eisinger, M.D.

For further information on registration, faculty, and fees, please contact:

Deborah Gray
Academy of Medicine of New Jersey
Two Princess Road,
Lawrenceville, New Jersey 08648
Phone: (609) 896-1717

ANNUAL CME ASSEMBLY

presented by the

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STATE OF NEW YORK

MAINTAINING THE QUALITY OF HEALTH CARE INTO THE 21st CENTURY

MARCH 7-9, 1986

THE NEW YORK HILTON, NEW YORK CITY

☐

HIGHLIGHTS

22 CME Sessions; Panel Discussions; Technical Exhibits: two Receptions sponsored by MSSNY: President's Dinner Dance.

Registration: There is no fee for members of the New Jersey State Medical Society, residents, medical students, or medical assistants. Nonmember physicians—$50; Allied Professions $10.

☐

For a copy of the preliminary program, contact: MSSNY, 420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Attn: Division of Meeting Services

516-488-6100

THE LEGACY OF KAREN ANN QUINLAN A TENTH YEAR RETROSPECTIVE.

Wednesday, March 26, 1986
8:30 A.M.-4:00 P.M.

RUTGERS MEDICAL SCHOOL
PISCATAWAY, NJ (Main Auditorium)

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AMERICAN COLLEGE OF PHYSICIANS, NEW JERSEY CHAPTER
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SOCIETY FOR HEALTH AND HUMAN VALUES
MEDICAL SOCIETY OF NEW JERSEY
UNIVERSITY OF MEDICINE AND DENTISTRY OF NEW JERSEY

Honorary Chairman: Thomas H. Kean, Governor of New Jersey

Panelists: George J. Annas, Christine K. Cassel MD, Eric J. Cassell MD, Hon. Richard J. Hughes, Joanne Lynn MD, Rev. John J. Paris, Mark Siegler MD, Alan J. Weisbard

Moderators: Paul Armstrong, Robert Cassidy, Russell L. McIntyre, Michael A. Nevins MD

Luncheon Speaker: Stanley S. Bergen MD

5 CME Category 1 Credits Approved

Registration $40.00 includes luncheon

For additional information contact Program Chairman:
Michael Nevins MD
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(201) 391-1078

Detach and mail to ACP, 4200 Pine Street, Philadelphia PA

Enclosed is my check for _____ payable to ACP

Name (please print) ____________________

Address ____________________

Phone ____________________

A confirmation will be sent to you along with a program and directions to Rutgers Medical School.

8 A.M.—Freehold Area Hospital
(AMNJ)

7 **Pediatric Infectious Disease**
14 **Rounds**
21 9-9:30 A.M.—Medical Science Bldg.,
28 B-610, NJ Medical School, Newark
(Newark Beth Israel Medical Center and AMNJ)

March

15 **Neonatal Surgical Emergencies**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

RADIOLOGY

February

20 **Radiology**
7:30-10 P.M.—Saint Barnabas Medical Center, Livingston
(Radiology Society of NJ and AMNJ)

27 **Visiting Professorship Program**
1:30-5 P.M.—Saint Barnabas Medical Center, Livingston
(Saint Barnabas Medical Center and AMNJ)

March

8 **Radiology of the GI Tract**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

SURGERY

February

4 **Surgical Grand Rounds**
18 7-9 A.M.—Hackensack Medical Center
(Hackensack Medical Center and AMNJ)

22 **Alternatives in Treatment of Abdominal Aortic Aneurysms**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

March

4 **Surgical Grand Rounds**
11 7-9 A.M.—Hackensack Medical
25 Center, Hackensack
(Hackensack Medical Center and AMNJ)

SURGICAL SPECIALTIES

February

8 **Care of the Postoperative Cardiac Surgery Patient**
8 A.M.-5 P.M.—Rutgers Medical School, Piscataway
(UMDNJ and AMNJ)

26 **Contributions to Vascular Surgery at Englewood Hospital**
8-10 P.M.—Englewood Club, Englewood
(Englewood Surgical Society and AMNJ)

March

15 **Neonatal Surgical Emergencies**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

EXECUTIVE OFFICES ☐ TWO PRINCESS ROAD, LAWRENCEVILLE, NEW JERSEY 08648 ☐ TELEPHONE 609-896-1766

DAILY SCHEDULE

220th Annual Meeting
Medical Society of New Jersey

Wednesday, May 14, 1986

3:30 p.m.— Board of Trustees' Meeting

Thursday, May 15, 1986

7:30 a.m.— Registration Opens
9:00 a.m.— Message Center and Exhibits Open
2:00 p.m.— House of Delegates
4:00 p.m.— Reference Committees

Friday, May 16, 1986

8:00 a.m.— Registration, Message Center, and Exhibits Open
9:00 a.m.— House of Delegates (Election)
12:00 noon— Golden Merit Award Ceremony
2:30 p.m.— Reference Committees
5:00 p.m.— JEMPAC Political Forum
5:45 p.m.— JEMPAC Wine & Cheese Reception

Saturday, May 17, 1986

8:00 a.m.— Registration and Message Center Open
8:30 a.m.— House of Delegates
1:30 p.m.— House of Delegates
6:30 p.m.— Inaugural Reception followed by Inaugural Dinner

Sunday, May 18, 1986

8:00 a.m.— Registration and Message Center Open
8:30 a.m.— Program of Major Interest to Physicians
1:00 p.m.— Board of Trustees' Meeting

The Westin Bellevue Stratford
Philadelphia, PA

ACUPUNCTURE IN CLINICAL PRACTICE

N.Y. State Boards of Medicine & Dentistry 25-hour accredited seminar and workshop on the latest theories & techniques of manual & electro-acupuncture and TENS, applicable toward the 200-hour requirement for certification, will be given for licensed clinicians (with or without prior training) during weekend, Jan. 10-12, 1986, Fri-Sat-Sun., Mulford Plaza Hotel, Manhattan & again weekend Mar 7-8-9, 1986, New York Pental Hotel, Manhattan. Co-sponsored by the International College of Acupuncture & Electro-Therapeutics, its official journal, Acupuncture & Electro-Therap. Res., Int.J. (published by Pergamon Press and Indexed in 15 major indexing periodicals; INDEX MEDICUS, etc.), the Heart Disease Research Foundation and the Neuroscience Dept. of Long Island College Hospital, Pharmacology Dept. of The Chicago Medical School. Also eligible for AMA/CME credit. For information, contact Y. Omura, M.D., ScD., 800 Riverside Drive (8-1), NYC 10032. Tel: (212) 781-6262 or (212) WA8-0658, Phil Shinick, Ph.D., Tel: (201) 246-8557.

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of
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presents

PREP FOR PRACTICE

A PRACTICE MANAGEMENT SEMINAR FOR RESIDENTS
SATURDAY, MARCH 1, 1986
9:00 A.M.-4:30 P.M.
at the Executive Offices of the
MEDICAL SOCIETY OF NEW JERSEY
Lawrenceville, New Jersey 08648

MORNING PROGRAM

9:00-9:15	Introduction
9:15-10:00	**Search for the Right Opportunity** • How the Changing Complexion of Health Care Will Affect You • Professional Consideration in Selecting a Site • Personal Considerations • Financial Considerations
10:00-10:15	Questions and Answers
10:15-11:15	**Types of Practice** • Solo • Partnership • Group • HMOs
11:15-11:30	Questions and Answers
11:30-12:00	**Physical Facilities** • Space Requirements • Floor Plans • Patient and Work Flow
12:00-12:15	Questions and Answers
12:15-1:00	Lunch

AFTERNOON PROGRAM

1:00-1:30	**Business Insurance** • Types. • How Much and When
1:30-1:45	Questions and Answers
1:45-2:30	**Personnel** • What You Need • How to Find • Job Descriptions • Performance Reviews and Salary Reviews
2:30-2:45	Questions and Answers

2:45-3:15	**Systems**
	• Time Management
	• Charts and Records
	• Phones
	• Billing, Insurance and Collection
	  • Fees
	  • Medicare

3:15-3:30	Questions and Answers
3:30-4:15	**Financial Management**
4:15-4:30	Questions and Answers
4:30	Adjournment

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LETTER TO THE EDITOR

Emergency Department Treatment

October 14, 1985

Dear Doctor Krosnick:

I read Doctor William A. Dwyer's article, "Method for Evaluating Hospital Emergency Department Treatment" (*J Med Soc NJ* 82: 523-528, 1985). As an emergency physician and the administrator of an emergency physician group, I found it interesting. However, there are a few statements, assumptions, and implications which, in my experience, are questionable.

As regards materials and methods:

1. Whenever the necessity of an emergency department visit is questioned, it invariably is answered retrospectively by trained health care personnel. Unfortunately, patients usually are not as medically sophisticated as the reviewers. And even physicians recognize that there are prospective signs and symptoms which must be evaluated immediately (regardless of the final impression) because they represent significant risk.
2. Whenever the quality of patient care is examined scientifically, the following conditions should exist: defined criteria determined prospectively, and appropriately experienced case appraisers. In this article, criteria are not defined; it appears that they were not prospectively determined, and the emergency medicine expertise of the physician reviewers is not mentioned.

My major reservations, however, concern the assertions and assumptions contained in the discussion and summary. For example, characterizing the emergency department patient throughput time as "pedestrian" is a little bit pejorative. In fact, the results show that more than 70 percent of the patients were discharged within 1-1/2 hours. Is this really such a terrible record considering the fact that the emergency department does not have the luxury of being able to schedule patients or to turn patients away? Nor does the emergency department usually have significant control over ancillary hospital services. I am sure that many patients consider a 1 to 1-1/2 hour throughput time in their personal physician's office (a selective, scheduled, and much more controlled environment) fairly routine. Also, it should be remembered that most physician's offices do not have the wide selection of diagnostics or the immediacy of result reporting which is available to the emergency department. Therefore, some of this time is used to better define the patient's condition.

The main implication seems to be: decreasing the number of nonemergent visits to the emergency department will save a substantial amount of money and positively affect quality of care. This is an attractive but highly simplistic premise.

The stated stimulus for undertaking this review was Medicaid's "... need to evaluate carefully the expenditure of health care dollars ..." so that the focus is economic. In any study it always is important to insulate the results from prestudy presumptions. This is especially true of retrospective reviews which by their very nature are highly sensitive to the reviewer's judgment.

The costs associated with maintaining an emergency department are dependent upon the interrelationship of three basic factors: the types (delineated by severity of illness or injury) of patients to be treated, the volume of patients in each severity classification, and the quality of care deemed acceptable for each patient within a specific classification. If we do not define quality of care but assume that it should be "good" and further assume that it would remain constant, then we can more easily examine the effects of volume upon costs.

Fundamentally, costs depend upon the type and amount of technology, i.e. space, diagnostics, instrumentation, and the amount of labor, i.e. personnel to operate the technology and provide direct care necessary to evaluate and treat a patient.

There is a direct relationship between the severity of a patient's medical condition and the amount of resources required. Patients with the most serious illnesses or injuries will need substantial technological support and a high personnel/patient ratio. Patients with the least serious illnesses or injuries require minimal technology and a low personnel/patient ratio.

What will happen if patients with the least serious illnesses or injuries are shifted away from the emergency department? If an emergency department must have the capability for evaluating and treating all levels of illness/injury severity (and that is the present philosophy), then the major cost determinants are the resources required by the most serious conditions.

Technology must be purchased and maintained and, since there is no way to schedule these types of visits, appropriate staffing is required 24 hours a day. Given this circumstance, a decrease in the number of patients with less serious medical conditions will not result in a large decrease in the emergency department's expense base, i.e. the decrease in the annual costs will not be proportional to the decrease in annual patient visits. There probably will be some decrease in costs, but the average cost per patient will rise for the remaining patient volume. This will result in an increased patient charge.

Of course, we can hypothesize a future where emergency medical care is stratified so that only a few hospitals will be designated to evaluate and treat the most serious emergency illnesses and injuries. But what happens then? The "lesser" emergency departments will not have to maintain the "high acuity care" cost items and, therefore, their expenses will decrease. The average cost per patient then should decrease significantly. However, this decrease will be partly offset by an increase in the average patient cost engendered by the siphoning off of patients with less serious medical conditions. In the "high acuity" emergency departments, the volume of patients with serious illnesses or injuries will increase, thereby producing an increased requirement for technology and labor. This, in turn, will produce increased costs for these institutions. I believe that the net expense decrease of all this shifting will be negligible, but even if it could be a moderate amount, there are some additional aspects of the hypothetical system which would have to be addressed:

1. The establishment and maintenance of a medically sophisticated screening process to insure that patients are directed to the appropriate type of facility. The additional expense of this system must be reckoned in the total cost.

2. The establishment and maintenance of a highly responsive patient transfer organization so that patients who have been incorrectly directed or whose medical condition has deteriorated could be quickly transported to an appropriate facility. The additional expense of this system must be reckoned in the total cost.

3. The possibility that the government might incur liability by mandating such a system. The possible costs of judgments must be reckoned.

Besides all of the above expense shifting, in both the real and hypothetical instances, there would be the additional costs of maintaining a nonhospital health care system which will treat patients with less serious illnesses or injuries and which must be responsive 24 hours a day, seven days a week.

Next, the issue of quality of care should be examined. The assumption that emergency department patient volume substantially impacts, over the long term, upon quality of care is specious. First, a prospective definition of what constitutes quality of care must be made. Second, the experience and knowledge of any appraiser must be appropriate for judging if a specific case fails to satisfy the definition. Conceding these two points, the fact is that quality of care is most dependent upon the experience, expertise, and effectiveness of the health care professionals providing the service. It is incumbent upon the hospital administration to insure that the number, training, and knowledge of the emergency department personnel (i.e. physicians, nurses) and the interface between the emergency department and other hospital areas are appropriate for the maintenance of good care at the prevalent level of patient visits. If the emergency department volume increases, then the revenue generated will increase (assuming the present outpatient reimbursement system remains in effect). I hope that hospital administrators would recognize the dangers of false economy and use some of this increased revenue to appropriately expand the emergency department's physical plant and staffing. I am more confident that if the volume were to decrease, resources (now viewed as "underutilized") would be trimmed. Therefore, quality of care in the emergency department ultimately is determined by hospital administration's perception of what constitutes an appropriate amount of money and energy to invest in the department.

The present emergency medical system has evolved from an interplay of a patient's need for easily accessible care and the demand upon hospitals to provide round-the-clock "high acuity" care. Any change in the present system must be approached cautiously and the sociological, economic, and legal consequences carefully evaluated.

(signed) Peter L. Sawchuk, M.D.
Emergency Medical Associates

BOOK REVIEWS

Atlas of Echocardiography; CAPD in Children; Illustrated Encyclopedia of Dermatology

Atlas of Echocardiography

Ernesto Salcedo, M.D. Philadelphia, PA, W.B. Saunders Company, 1985. Pp. 403. ($45)

This revised second edition represents a most comprehensive review of echocardiography.

Chapter one presents tomographic anatomy of the heart. Through multiple diagrams, the reader is conditioned to see the heart as a truly three-dimensional structure which must be viewed in two-dimensional slices. Definitions of the standard planes used for reference are given.

In chapter two, the manipulation of the transducer to obtain these tomographic views is discussed. Standard measurements obtained by M mode and 2-D echocardiography are summarized. Chapter three illustrates the relationship between phonocardiography and echocardiography. Subsequent chapters then are devoted to specific anatomic subdivision of the heart, e.g. mitral valve, aortic valve, aorta, and left and right ventricle. Three separate chapters are reserved to illustrate prosthetic valves, coronary artery disease, and infective endocarditis. Chapter 15 covers diseases of the pericardium with sections on effusion, tamponade, constriction, and metastatic disease. The last chapter focuses on congenital heart disease and presents a most exhaustive review with diagrams and reproductions of M mode and 2-D echocardiograms.

The diagrams used throughout this book are well done. The photographic reproductions are first quality. Captions are complete and concise. The text is on glossy paper which may produce glare with some light sources but is necessary to achieve high resolution photoreproductions.

This book must be recommended as a reference for the student of echocardiography at any level. It most properly belongs next to each machine for quick consultations.

Robert M. MacMillan, M.D.

CAPD in Children

R.N. Fine, K. Scharer, O. Mehls (eds). New York, NY, Springer-Verlag 1985. Pp. 220. Illustrated. ($24.50)

Continuous ambulatory peritoneal dialysis (CAPD) was described in 1976, and became a clinically applicable procedure in pediatrics in 1980. Since that time, it appears to have become a preferred method of treating children with end-stage renal disease.

This monograph on CAPD is composed of the papers presented at the First International Symposium on CAPD in Children, which was held in May 1984. As such, it contains discussions of a potpourri of topics, including physiology, pathophysiology, and dialysis technique, based on the research and experience of the symposium participants. Many of the articles present only preliminary data, or results whose validity might be questioned due to the small numbers of patients investigated, the short followup periods, or results at variance with previously reported, though similarly flawed, studies. These problems are unavoidable, considering that CAPD as a therapeutic modality still is in its infancy. At the same time, the limited experience generally available makes some articles especially useful, e.g. "The Management of the Infant on CAPD." The articles are clearly written with adequate accompanying tables and figures.

CAPD in Children is not a textbook of pediatric CAPD, as its name might imply. Its contents will be of interest mainly to those doing research involving CAPD, and occasionally to those facing specific problems in patient management.

Howard W. Rosenblum, M.D.

Illustrated Encyclopedia of Dermatology, Second Edition

Lionel Fry, M.D., Fenella T. Wojnarowska, M.D., Parvin Shahrad, M.D. Oradell, NJ, Medical Economics Books, 1985. Pp 575. Illustrated. ($29.95)

This second edition of *Illustrated Encyclopedia of Dermatology* encompasses the past four years; the text is as close to current thought as possible. It is authored and printed in Great Britain and enclosed in a "casebound" cover.

Although many conditions are arranged alphabetically, some are grouped under a category, i.e. "Deficiency states," "Genodermatoses," necessitating a perusal of the index in order to read the specific entity desired.

The material is presented in a concise and delineated fashion and meets the requirements of an encyclopedia. All illustrations are in black and white and leave much to be desired in that aspect.

The treatment section varies widely: for some conditions, merely a line, while others are more detailed and current.

The book is satisfactory as a quick reference and its listed price is not expensive.

Murray Kahn, M.D.

OBITUARIES

Drs. Berman; Fortuin; Hartmann; Lyons; Magee; Munro; Smith; Teller

Dr. Jacob J. Berman

A retired family practitioner in Trenton, Jacob Julius Berman, M.D., died on September 5, 1985, at home, at the advanced age of 86. Born in New Haven, Connecticut, Dr. Berman was graduated from Jefferson Medical College, Philadelphia, in 1919, and became a member of our Mercer County component, and of the American Medical Association. Prior to his retirement, Dr. Berman had been affiliated with Mercer Medical Center, Trenton. In 1969, in recognition of his 50th anniversary as a physician, Dr. Berman received MSNJ's Golden Merit Award.

Dr. Floyd Fortuin

A member of our Passaic County component, Floyd Fortuin, M.D., died on August 27, 1985, at the age of 76. A native of Paterson, Dr. Fortuin received his medical degree from Cornell University Medical College, in 1935. He established his practice in psychiatry and neurology in the city of his birth, and was affiliated with St. Joseph's Hospital and Medical Center in that city, as well as Valley Hospital, Ridgewood, and Chilton Memorial Hospital, Pompton Plains. He was board certified in his specialty, and had served in the medical corps of the United States Air Force during World War II, emerging with the rank of major. At last year's Annual Meeting, Dr. Fortuin was one of the recipients of MSNJ's Golden Merit Award honoring his 50 years in medicine.

Dr. Edmund M. Hartmann

An industrial physician, affiliated with the American Cyanamid Company for most of his career, Edmund Michael Hartmann, M.D., died on August 2, 1985. Born in New York City, in 1912, Dr. Hartmann was graduated from St. Louis University School of Medicine in 1939. He had been a member of our Somerset County component, but finally joined the Union County Medical Society when his place of employment changed to the Linden office. From 1941 to 1946, Dr. Hartmann was in the medical department of the United States Army, with the rank of major.

Dr. Romola Lyons

At the remarkable age of 101, retired dermatologist, Romola Lyons, M.D., died at the Putnam Hospital Center, Carmel, New York, on August 2, 1985. Born in Brooklyn, Dr. Lyons obtained her medical degree from Columbia University College of Physicians and Surgeons in 1929. She was a member of our Bergen County Component and of the American Medical Association. Dr. Lyons had been on the staff at Vanderbilt Clinic, New York City, and Englewood Hospital. In 1979, Dr. Lyons was a recipient of MSNJ's Golden Merit Award in recognition of her 50th year in medicine.

Dr. Harold S. Magee

A retired psychiatrist and neurologist, Harold Sinclair Magee, M.D., died at North Ridge General Hospital, Fort Lauderdale, Florida, on August 23, 1985, at the venerable age of 91. Born in London, Ontario Province, Canada, Dr. Magee earned his medical degree at the University of Western Ontario, Canada, in 1925. For many years he served as medical director at Trenton Psychiatric Hospital. Dr. Magee was a member of our Mercer County component and of the American Medical Association. He was board certified in his specialty. In 1975, Dr. Magee received the Medical Society's Golden Merit Award, honoring his 50 years as a physician.

Dr. Jeannette Munro

Princeton's first pediatrician, Jeannette Munro, M.D., died in Nyack, New York, on August 12, 1985, in her 92nd year. A native of Philadelphia, Dr. Munro was graduated from the University of Wisconsin's Medical School in 1927. She first practiced her specialty in New York, but moved to Princeton to establish an office in 1934, where she continued to practice until her retirement in 1964. Dr. Munro was a member of our Mercer County component, and of the American Medical Association. She was board certified in her specialty. In 1977, Dr. Munro was honored with MSNJ's Golden Merit Award, marking her 50 years in the medical profession.

Dr. Ellis L. Smith

Ellis Leon Smith, M.D., a retired member of our Essex County component, died at the Troy Hills Center, Parsippany, on August 9, 1985, after a long illness. Dr. Smith, born in 1898, and a native of Texas, received his medical degree from Baylor University College of Medicine, Houston, in 1922. He had served as medical director at the Essex County Isolation Hospital, Belleville, for 35 years prior to his retirement to Colorado in 1971. In 1972, MSNJ conferred its Golden Merit Award on Dr. Smith in recognition of his 50 years as a physician.

Dr. Daniel W. Teller

A Morristown family practitioner, Daniel Woolsey Teller, M.D., died on August 25, 1985, at the venerable age of 84. A native of Westwood, Dr. Teller was graduated from Temple University School of Medicine, Philadelphia, in 1928. He was a member of our Morris County component and of the American Medical Association. He had been affiliated with Morristown Memorial Hospital, and was chief of pediatrics at All Souls Hospital, Morristown. Dr. Teller had served as medical director of the Red Cross' disaster relief program, and of the well-baby clinics in Morristown. In 1978, Dr. Teller was a recipient of MSNJ's Golden Merit Award, honoring his 50 years in medicine.

AUTHOR INFORMATION

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CONTENT

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2. Dixon WJ, Massey FJ: *Introduction to Statistical Analysis.* New York, NY, McGraw-Hill, 1969, pp. 42-48.

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WARNINGS: FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS.

BACTRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION. Clinical signs, such as rash, sore throat, fever, pallor, purpura or jaundice, may be early indications of serious reactions. In rare instances a skin rash may be followed by more severe reactions, such as Stevens-Johnson syndrome, toxic epidermal necrolysis, hepatic necrosis or serious blood disorder. Perform complete blood counts frequently.

BACTRIM SHOULD NOT BE USED IN THE TREATMENT OF STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have a greater incidence of bacteriologic failure when treated with Bactrim than with penicillin.

PRECAUTIONS:

General: Give with caution to patients with impaired renal or hepatic function, possible folate deficiency and severe allergy or bronchial asthma. In glucose-6-phosphate dehydrogenase-deficient individuals, hemolysis may occur, frequently dose-related.

Information for Patients: Instruct patients to maintain adequate fluid intake to prevent crystalluria and stone formation.

Laboratory Tests: Perform complete blood counts frequently; if a significant reduction in the count of any formed blood element is noted, discontinue Bactrim. Perform urinalyses with careful microscopic examination and renal function tests during therapy, particularly for patients with impaired renal function.

Drug Interactions: In elderly patients concurrently receiving certain diuretics, primarily thiazides, an increased incidence of thrombocytopenia with purpura has been reported. Bactrim may prolong the prothrombin time in patients who are receiving the anticoagulant warfarin. Keep this in mind when Bactrim is given to patients already on anticoagulant therapy and reassess coagulation time. Bactrim may inhibit the hepatic metabolism of phenytoin. Given at a common clinical dosage, it increased the phenytoin half-life by 39% and decreased the phenytoin metabolic clearance rate by 27%. When giving these drugs concurrently, be alert for possible excessive phenytoin effect. Sulfonamides can displace methotrexate from plasma protein binding sites, thus increasing free methotrexate concentrations.

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Carcinogenesis, Mutagenesis, Impairment of Fertility: Carcinogenesis: Long-term studies in animals to evaluate carcinogenic potential not conducted with Bactrim. *Mutagenesis:* Bacterial mutagenic studies not performed with sulfamethoxazole and trimethoprim in combination. Trimethoprim demonstrated to be nonmutagenic in the Ames assay. No chromosomal damage observed in human leukocytes *in vitro* with sulfamethoxazole and trimethoprim alone or in combination; concentrations used exceeded blood levels of these compounds following therapy with Bactrim. Observations of leukocytes obtained from patients treated with Bactrim revealed no chromosomal abnormalities. *Impairment of Fertility:* No adverse effects on fertility or general reproductive performance observed in rats given oral dosages as high as 70 mg/kg/day trimethoprim plus 350 mg/kg/day sulfamethoxazole.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Trimethoprim and sulfamethoxazole may interfere with folic acid metabolism; use during pregnancy only if potential benefit justifies potential risk to fetus. Nonteratogenic Effects: See CONTRAINDICATIONS section.

Nursing Mothers: See CONTRAINDICATIONS section.

Pediatric Use: Not recommended for infants under two months (see INDICATIONS and CONTRAINDICATIONS sections).

ADVERSE REACTIONS: Most common are gastrointestinal disturbances (nausea, vomiting, anorexia) and allergic skin reactions (such as rash and urticaria). **FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS (SEE WARNINGS SECTION).** *Hematologic:* Agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, neutropenia, hemolytic anemia, megaloblastic anemia, hypoprothrombinemia, methemoglobinemia, eosinophilia. *Allergic Reactions:* Stevens-Johnson syndrome, toxic epidermal necrolysis, anaphylaxis, allergic myocarditis, erythema multiforme, exfoliative dermatitis, angioedema, drug fever, chills, Henoch-Schoenlein purpura, serum sickness-like syndrome, generalized allergic reactions, generalized skin eruptions, photosensitivity, conjunctival and scleral injection, pruritus, urticaria and rash. Periarteritis nodosa and systemic lupus erythematosus have been reported. *Gastrointestinal:* Hepatitis (including cholestatic jaundice and hepatic necrosis), elevation of serum transaminase and bilirubin, pseudomembranous enterocolitis, pancreatitis, stomatitis, glossitis, nausea, emesis, abdominal pain, diarrhea, anorexia. *Genitourinary:* Renal failure, interstitial nephritis, BUN and serum creatinine elevation, toxic nephrosis with oliguria and anuria, crystalluria. *Neurologic:* Aseptic meningitis, convulsions, peripheral neuritis, ataxia, vertigo, tinnitus, headache. *Psychiatric:* Hallucinations, depression, apathy, nervousness. *Endocrine:* Sulfonamides bear certain chemical similarities to some goitrogens, diuretics (acetazolamide and the thiazides) and oral hypoglycemic agents; cross-sensitivity may exist. Diuresis and hypoglycemia have occurred rarely in patients receiving sulfonamides. *Musculoskeletal:* Arthralgia, myalgia. *Miscellaneous:* Weakness, fatigue, insomnia.

DOSAGE AND ADMINISTRATION: Not recommended for use in infants less than two months of age.

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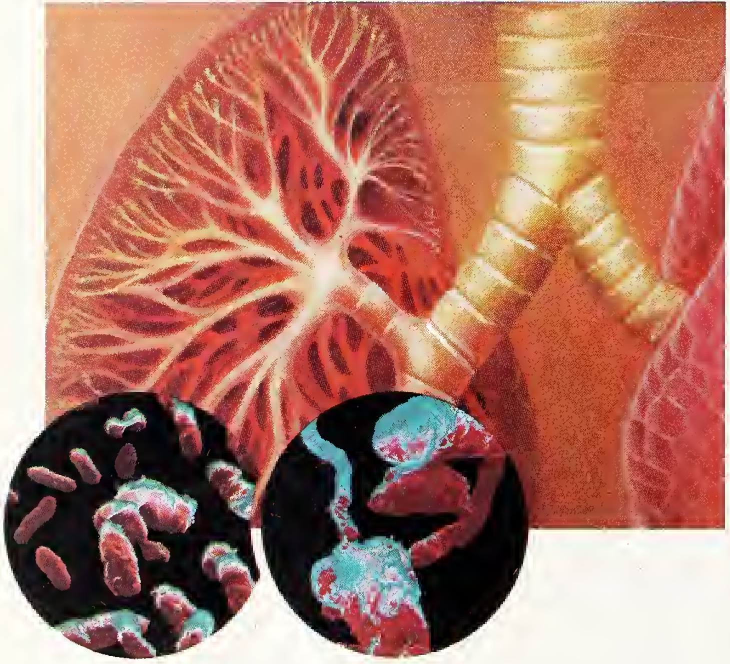

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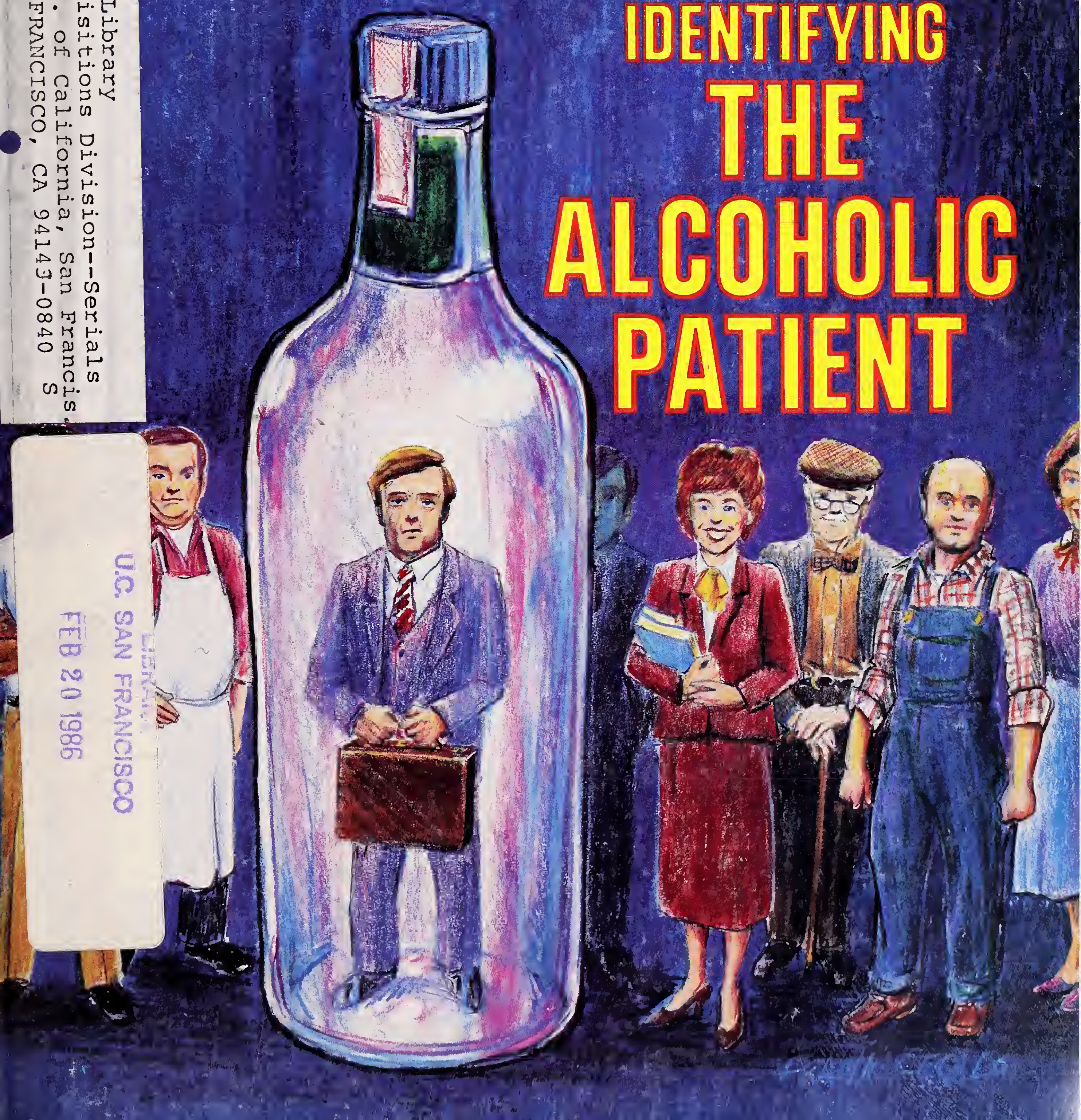

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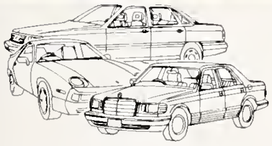

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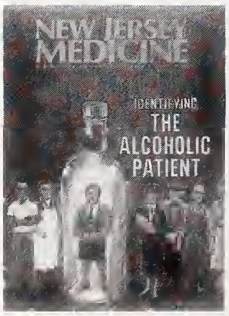
On The Cover: Physicians are confronted by alcoholic patients and need a diagnostic game plan to deal with such patients. Our cover story begins on page 87. Cover illustration by Frank Cecala.



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MEMBERSHIP NEWSLETTER

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THE MEDICAL SOCIETY OF NEW JERSEY VOLUME 30

OVERDUE MEDICAL PAYMENTS UNDER NO-FAULT IN NEW JERSEY

Our no-fault statute provides that personal injury protection benefits are overdue if not paid within 30 days after the carrier receives written notice of the claim. Overdue payments bear interest at the rate prescribed in the Rules of Court. That rate currently is 12 percent per annum.

A claim is not overdue if the carrier supplies written notice of denial. A carrier also may request a 45-day extension for further investigation; the request must be in writing and must specify the reasons for the extension.

All claimants have the right to proceed to arbitration. If the claimant is successful, the carrier is to pay all costs including attorney fees.

Don't let carriers arbitrarily withhold medical payments. If you are experiencing difficulty, contact Joseph Lucci at the Medical Society of New Jersey.

CHILD ABUSE REPORTING

It is the law in New Jersey that any person having reasonable cause to believe that a child has been subjected to abuse shall report this promptly to the Division of Youth and Family Services of the Department of Human Services.

The toll-free number for reporting is 800-792-8610.

No one is exempt from this responsibility. Further, a knowing violation subjects one to being charged as a disorderly person. This law has been in effect since 1972 and is in accord with the rest of the states, all of which have some kind of reporting statute.

The reporting of acts of child abuse is increasing astronomically, and so all citizens should do their part to prevent the abuse of those who are least able to protect themselves.

STOP SMOKING CAMPAIGN

Doctors, who are viewed as role models and whose advice usually is taken seriously, can help a majority of smokers quit, according to internists. As the nation's major providers of continuing, comprehensive care to adolescents and adults, internists see the costly consequences—both in dollars and lives—of smoking-related diseases each day. As a result, the American Society of Internal Medicine (ASIM) launched a campaign on October 10, 1985, during its 29th Annual Meeting in Washington, D.C., to call upon all physicians, regardless of specialty, to quit smoking and to prohibit smoking in their offices. In addition, ASIM is urging physicians to begin or expand patient education programs on smoking cessation.

Citing studies showing that 1) 85 percent of smokers want to quit; 2) physicians hold the most influence in helping patients quit; and 3) 38 million of the 54 million smokers will see a physician at least once a year, ASIM has developed a kit of materials to help physicians establish a smoke-free office environment. A poster and tent card warn patients that "When It Comes To Good Health, Don't Let Yours Go Up In Smoke." A "No Smoking" lapel pin, tip sheet, and list for patient education materials are included.

Kits can be ordered by sending $5 (members) or $8 (nonmembers) for each to ASIM STOP SMOKING CAMPAIGN, 1101 Vermont Ave., NW, #500, Washington, DC 20005.

IMPAIRED PHYSICIANS PROGRAM

Statistics for the first three years of the Impaired Physicians Program present a physician-client census of 259 physicians. It is significant that of these physicians referred to the program, 56 percent have remained clinically well and free of their presenting problem from their initial contact with the Program. We encourage those who know of a physician who may need the services of the Program to contact the Program staff in Doctor Canavan's office at the Medical Society of New Jersey, (609) 896-1766.

BE CAREFUL

The Drug Utilization Review Council of the New Jersey Department of Health on December 10, 1985, indicated approval of substitute generic equivalent tablets of "warfarin sodium" to the list of interchangeable drugs.

Patients on this potent drug require frequent monitoring and switching brands may necessitate additional prothrombin-time tests.

Physicians may want to restrict the trial of generic equivalent warfarin sodium to new patients.

The availability of substitutes began in January 1986. You still may indicate your no-substitute privilege if you so desire.

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PROFESSIONAL LIABILITY COMMENTARY*

Reducing Malpractice Risks

ER Malpractice; Alternative Practice Arrangements; Malpractice Award to NJ Patient; Did You Know; A More Balanced Medical Liability System; Attorney's Fee

ER MALPRACTICE RISK COULD BE CUT IN HALF

Physicians undoubtedly agree—even though many lawyers might not—that with malpractice suits, as with disease, prevention is infinitely preferable to attempts to cure. Unhappily, no one has discovered a vaccine that confers lifetime immunity to litigation.

And doctors who never have been sued hardly need to be reminded that they nevertheless pay for the sins, real or fancied, of those who have. Dr. John T. Rogers still has the malpractice policy he bought in 1954. It covered him for $100,000, with a $300,000 maximum yearly exposure, for an annual premium of $103. The same coverage today in the same state—with no record of litigation and no change in type of practice—costs $28,000. Says Dr. Rogers, "When one physician is sued, bear in mind that we're all sued. Indirectly, none of us is immune, even though we never may have the misfortune of being a defendant."

If there is no immunity, there still is a great deal that can be done to reduce the risk of suit, as Dr. Rogers points out in *Risk Management in Emergency Medicine*, published by the American College of Emergency Physicians. Certified in surgery and ob-gyn, Dr. Rogers holds masters' degrees in mathematics and actuarial science and has served as a malpractice claims examiner for eight insurance companies and teaches medical jurisprudence at Stetson University in DeLand and at the University of South Florida in Tampa.

Conscientious risk management can have a real impact on the frequency and the cost of litigation, Dr. Rogers contends. He points out that just five categories of cases—extremity fractures, a foreign body in wounds, myocardial infarction, laceration of tendons or nerves, and meningitis—are responsible for more than two-thirds of emergency medicine suits and costs. Reducing the number of lawsuits by half would thus reduce overall malpractice costs in that area by one-third. And, says Dr. Rogers, that can and must be done. His 36-page book contains many specific pointers on how to reduce the risk of a malpractice suit. He suggests putting yourself in the patient's place, strapped to a gurney and viewing the instruments and machinery from a horizontal position. It can be frightening, he says, and it is important to allay a patient's fear; a friendly attitude helps.

The physician in the emergency room is a stranger and may not even speak the same language. But assuming the patient can communicate, Dr. Rogers includes "rule-out" lists for complaints of chest pain, abdominal pain, and orthopedic trauma. There also is an insurance primer with invaluable advice plus general advice on what to do if you are sued.

Though the book is aimed at emergency physicians, it offers much that could be helpful to any doctor in regular practice. It is available from ACEP Distribution Center, P.O. Box 619911, Dallas, TX 75261-9911. (*Medical World News*, May 27, 1985)

ARE MDs IN ALTERNATIVE PRACTICE ARRANGEMENTS MORE LIKELY TO LOSE SUITS?

Cost-containment constraints built into Health Maintenance Organizations (HMOs), Preferred Provider Organizations (PPOs), and Independent Practice Associations (IPAs) suggest that physicians practicing in such arrangements are 20 to 35 percent more likely to lose malpractice suits, the Physicians Insurance Company of Michigan (PICOM) was told. Concerned about such possible increasing liability, the company asked three Michigan law firms to review their malpractice case experience and that was the consensus the lawyers reached. Why? Plaintiffs' lawyers could argue that patients in traditional practices would have received greater intensity of medical services. A PICOM plan to surcharge such physicians has been withdrawn, pending further study. "We have no quarrel with cost containment," Brian Hodge, PICOM communications spokesman, told *MLM*, "but we are concerned that this heightened liability will generate increased litigation and insurance costs." (*Medical Liability Monitor*, Volume 10, Number 9, September 27, 1985)

MALPRACTICE AWARD TO 72-YEAR-OLD PLAINTIFF

A $344,333 jury verdict has been reached in a malpractice suit brought by a 72-year-old man against a New Jersey hospital and one of its nurses. The plaintiff sued the hospital and nurse claiming he suffered permanent damage to his right arm when an intravenous needle was administered improperly, according to the plaintiff's attorney. The plaintiff's attorney said his client was receiving chemotherapy treatments as an outpatient at the hospital when the incident occurred on May 28, 1980. He said the nurse injected a chemotherapy fluid into the plaintiff's arm, missed the vein, and flooded surrounding soft tissue, damaging muscles and nerves. A one-day trial in Elizabeth resulted in a jury awarding the plaintiff $344,333 and $68,866 to his wife. (*Newark Star Ledger*, May 10, 1985)

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*This item from the Department of Professional Liability Control, MSNJ, was prepared by James E. George, M.D., J.D., and A. Ronald Rouse, who are, respectively, Director of the Department and Director of Special Projects.

DID YOU KNOW . . .

A proposed law that would permit Armed Forces personnel to sue the U.S. government for alleged medical malpractice acts should not be enacted, officials of Justice and Defense Departments testified before a House subcommittee this month? Such legislation "would cause a flood of litigation ... most of it vexacious and harassing," one Justice Department spokesman warned. (*Medical Liability Monitor*, Vol. 10, No. 7, July 31, 1985)

A MORE BALANCED MEDICAL LIABILITY SYSTEM

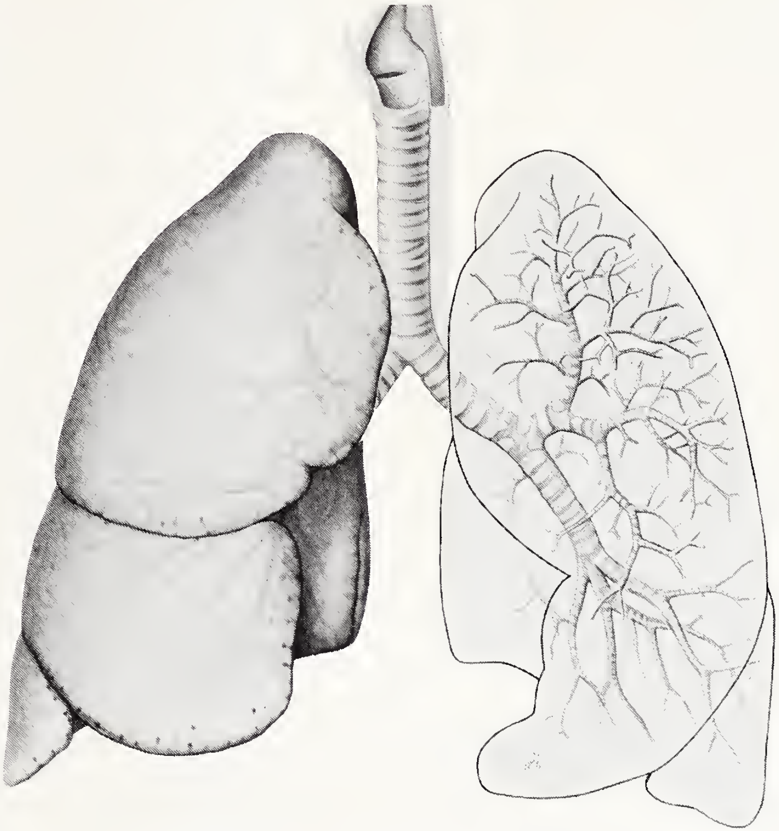
The September 1985 issue of *Nation's Business* contained the following paragraph in its "Where I Stand" portion of the magazine. "Many patients regard doctors as deities and think that modern medicine can cure everything. Such high expectations are leading juries to award big judgments in medical malpractice cases even when negligence has not been proved. The price of doctor's liability insurance is skyrocketing, and this is costing businesses plenty. Of the $100 billion spent yearly on employee health insurance, up to $14 billion goes for "defensive" medicine—unnecessary tests done as a defense against possible lawsuits—and to pay for physicians' malpractice coverage. Others disagree. Should we have a more balanced medical liability system?"

The response (November 1985 issue) to whether malpractice laws should be revised to make liability insurance less expensive for physicians and patients produced the following results: 89 percent responded, yes; 5 percent responded, no; and the remaining 6 percent were undecided.

COURT ALLOWS REQUESTED ATTORNEY'S FEE IN MALPRACTICE SUIT

An attorney who negotiated a settlement guaranteeing payments of almost $10 million to a severely mentally retarded child was entitled to an attorney's fee of $148,900.66, a New Jersey trial court ruled. The patient was born on July 29, 1978, and was severely mentally retarded. She also suffered from convulsions secondary to brain damage. A physician and hospital allegedly were negligent in failure to use available electronic fetal heart monitoring during labor. Just before trial a structured settlement was reached. The total settlement was $775,000. The minimum guaranteed payout under the settlement agreement was $9,707,544. The patient's attorneys filed a claim for a fee of $148,900.66. Allowing the claim, the court said that the request was not unreasonable. (*The Citation*, Vol. 51., No. 9, August 15, 1985)

Consider the causative organisms...



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Brief Summary Consult the package literature for prescribing information.

Indications and Usage: Ceclor* (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae)*, *Haemophilus influenzae*, and *S pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions: *General Precautions* – If an allergic reaction to Ceclor* (cefaclor, Lilly) occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e g., pressor amines, antihistamines, or corticosteroids

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest* tablets but not with Tes-Tape* (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy – Pregnancy Category B – Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor* (cefaclor, Lilly). There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers – Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor is administered to a nursing woman

Usage in Children – Safety and effectiveness of this product for use in infants less than one month of age have not been established

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy No serious sequelae have been reported Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain – Transitory abnormalities in clinical laboratory test results have been reported Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic – Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40)

Hematopoietic – Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal – Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

[061782R]

Note: Ceclor* (cefaclor, Lilly) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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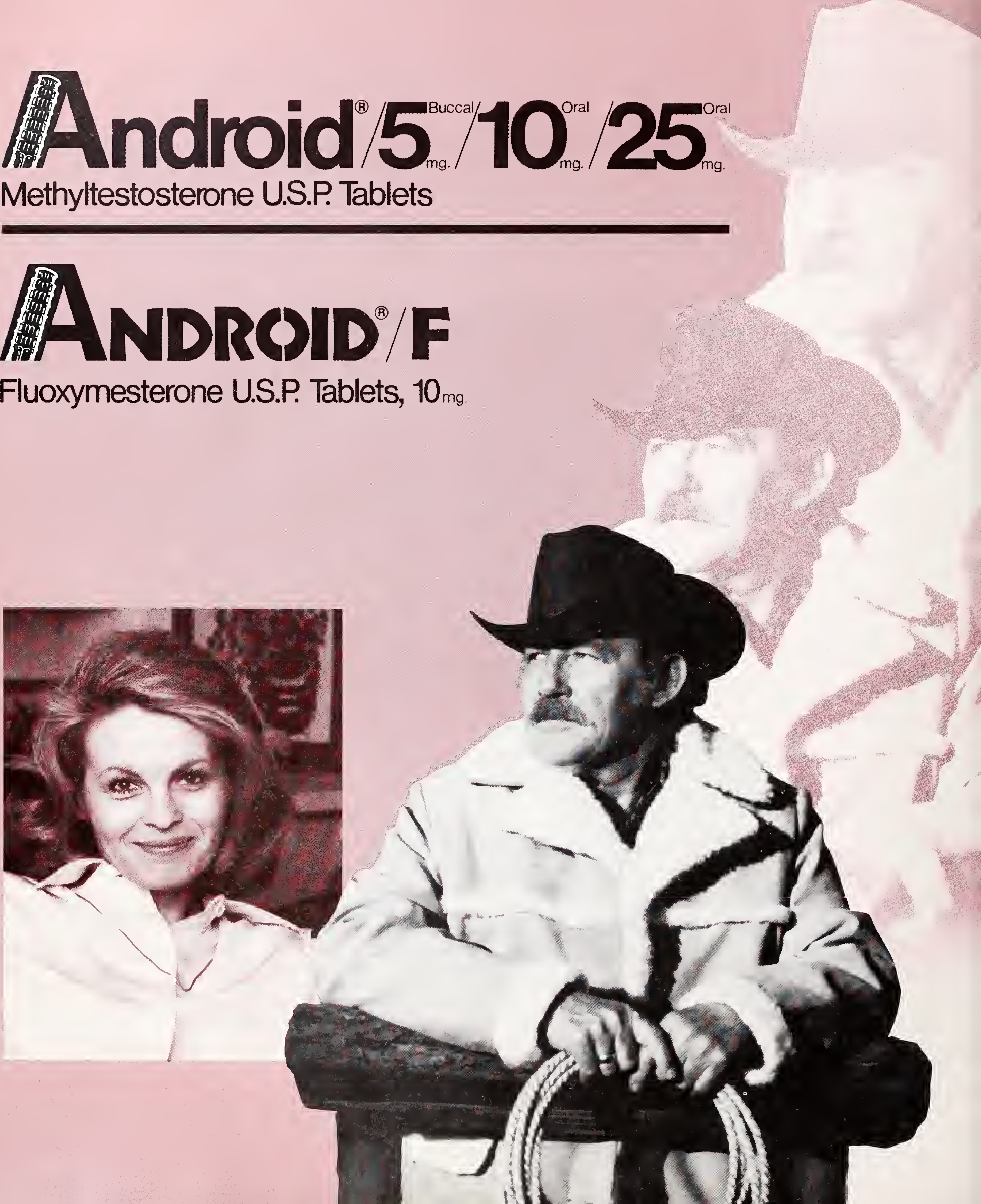

EDITORIAL

The Sammy Davis, Jr. National Liver Institute

A special center dedicated to the problems of liver disease has opened at UMDNJ-New Jersey Medical School, Newark.

We are all aware of the fact that liver disease is a major cause of morbidity and mortality. The causes of acute and chronic liver diseases are multiple, including infections, substance abuse, and other etiologies. In the United States, it is the fourth leading cause of death among men aged 40 to 59. The problem is attacking breadwinners at the peak of their careers, and young people whose lifestyles include illicit drug abuse, often in combination with alcohol. New Jersey's inner cities, such as Newark, have had more than their share of these problems.

While plagued with the problem of liver disease, our state has been most fortunate to have one of the world's experts in the pathogenesis, pathology, clinical manifestations, and treatment of alcoholic cirrhosis and other hepatopathies—Dr. Carroll M. Leevy, chairman of the department of medicine at the UMDNJ-New Jersey Medical School, Newark. Dr. Leevy, the recipient of many awards, has dedicated his scientific career to the study of the liver and its ailments.

What would be more appropriate than to establish a special center dedicated to the problem in New Jersey under the leadership of Dr. Leevy? That is exactly what was done on the campus in Newark through the help of the great entertainer, Sammy Davis, Jr.—a victim of liver disease. At a fundraising dinner, along with such luminaries as Bill Cosby, actor Anthony Quinn, actress Sally Struthers, rock artist Tommy Shaw, and former Miss America, Suzette Charles, Mr. Davis pledged his commitment and dedication to the Institute.

The National Liver Institute, an independent, nonprofit organization is thought to be "the world's first effort and resource devoted solely to patient care, education, and research for liver disorders." The Institute is temporarily housed at the medical school and the University Hospital until a freestanding building is erected. Future plans include a liver transplant surgical unit and diagnostic and treatment programs directed toward cancer of the liver.

We hope the Institute also will have time, space, and personnel to work on the social and psychological factors which are an important part of the problem, along with the appropriate scientific studies. In the meantime, all physicians should be aware of and support the Institute, a New Jersey and world first. A.K.

Practical and Legal Considerations: Collective Bargaining

VINCENT A. MARESSA, J.D.

This essay confronts the narrow issue of collective bargaining and whether physicians may engage in that activity.

Collective bargaining as contemplated by the National Labor Relations Act is a procedure looking toward the making of collective agreements between employer and accredited representatives of employees concerning wages, hours, and other conditions of employment. Collective bargaining requires that parties deal with each other with open and fair minds and sincerely endeavor to overcome obstacles existing between them to the end that employment relations may be stabilized and the obstruction to free flow of commerce prevented (National Labor Relations Act §8(5), 29 U.S.C.A. §158(5).

This definition is the law. Representatives of employees who have been certified by the National Labor Relations Board (NLRB) may engage in collective bargaining. They may organize strikes, picketing, slowdowns, and walk-outs without fear of prosecution for violation of state and federal antitrust laws.

Collective bargaining may only be engaged in by authorized unions. Without NLRB recognition, a clear antitrust conspiracy would exist.

MAY A PHYSICIAN UNION COLLECTIVELY BARGAIN?

A physician union can collectively bargain—but only if the members are employees within the context of the National Labor Relations Act and if their organization has been recognized by the NLRB. This means the physicians work for salary or wages at a site determined by the employer. The employer controls and directs services as to place, time, and detail. "Employee" must be distinguished from independent contractor, agent, or executive employee. The latter term means anyone who directs, manages, or supervises others. It has been used by the NLRB to deny recognition to an organization which filed to represent employed physicians (the National Federation of Physicians & Dentists). The NLRB denied the petition because the physicians had latitude in rendering professional services and served on various professional and administrative committees of the HMO where they were full time, salaried executive employees.

Consequently, it is evident that physicians in private practice are not employees within the National Labor Relations Act. They are independent contractors—not employees. The fact that they have joined what may be called a "union" is meaningless. That entity cannot bargain for them unless it has been certified by the NLRB. I am unaware of any such certification for any organization in New Jersey or on a national basis.

Physicians must remember it is not what the entity calls itself that matters—it is what it is authorized to do and what it actually does that must be evaluated.

MAY AN IPA COLLECTIVELY BARGAIN?

An Independent Physicians Association (IPA) may not collectively bargain—it is not an organization of employees but, by definition, is an association of independent practitioners, i.e. independent contractors. Therefore, it will not be granted the protective cloak of a "union" if it attempts to collectively bargain. IPAs present both opportunity and pitfalls for physicians. Under current antitrust theory, they should not represent all physicians within a given geographic area, they may not require members to join them exclusively, and they may not have the physician members control applicable fee schedules and negotiations with the HMO with which they contract.

The formation of an IPA does not grant that IPA territorial rights regarding a given HMO. The HMO will decide which IPA it will contract with and upon what terms. It can and often does form its own contracting IPA.

CONCLUSION

I sincerely hope this essay is of some value to the medical community. It is intended to confront the narrow issue of collective bargaining and whether physicians may engage in that activity. I do not believe they can under any circumstances that currently prevail in New Jersey.

In terms of representing physicians, the Medical Society of New Jersey has the capacity to negotiate, advise, and recommend on their behalf. Given the ingrained democratic concepts within the MSNJ structure, it is unrealistic to expect anything else!

The Medical Society
of New Jersey Auxiliary

TWO PRINCESS ROAD, LAWRENCEVILLE, NEW JERSEY 08648
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Dear Doctor:

The Medical Society of New Jersey Auxiliary is an active, vital organization whose accomplishments are many and varied. When our accomplishments have fallen short of our goals, we realize that the limitation of manpower often is the reason.

If your spouse is not a member of the Medical Society of New Jersey Auxiliary you both may be unaware of the Auxiliary's efforts on the state level to work for the passage of Medical Society of New Jersey-sponsored liability bills presently in the Senate. Do you know that the Auxiliary is sponsoring and establishing self-help groups for families suffering the stress of malpractice litigation? Are you both aware of the Impaired Spouse Committee which is working to help those among us who are in need?

These are but a few of the projects to which the Medical Society of New Jersey Auxiliary has committed itself. Your spouse can become better informed about these and other issues by becoming a member through your component medical society or by becoming a member-at-large of the State and National Auxiliaries.

I would appreciate your taking the time to discuss Auxiliary participation with your spouse. You both would benefit.

Please complete the form below and send it along with your check in the amount of $20 (payable to "MSNJ Auxiliary") to the Medical Society of New Jersey Auxiliary, Two Princess Road, Lawrenceville, New Jersey 08648. This will provide your spouse with all state and national publications to help be better informed of the issues important to the practice of medicine today. If you are a member of a County Medical Society where there is an active Auxiliary, your spouse will be contacted for County Auxiliary membership.

Thank you for taking the time to read this message. We are all working in the interest of medicine!

Very sincerely yours,

Mrs. Charles J. Moloney (Bernardine)
President

MEDICAL SOCIETY OF NEW JERSEY AUXILIARY

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HOSPITAL GOVERNING BOARDS*

An Unholy Alliance: The NJHA and the AFL-CIO

VINCENT A. MARESSA, J.D.

A proposed statewide network of 20 to 25 hospital-based HMOs sponsored by the AFL-CIO will affect hospitals and physicians in New Jersey.

Long and protracted negotiations have been conducted between the New Jersey Hospital Association (NJHA) and the state chapter of the AFL-CIO. Their purpose is to establish a statewide network of 20 to 25 hospital-based HMOs sponsored by the AFL-CIO. Newspaper accounts sporadically have touched upon the process but have not been very revealing. It was reported (September 1985) that the NJHA was advancing the sum of $250,000 to further the project.

I do not question the right of the NJHA to engage in HMO activity with the AFL-CIO—that is a matter to be determined by the trustees of the NJHA. What troubles me, however, is the callous disregard of that Association for the physicians of New Jersey and hospital medical staffs, in particular. Additionally, there is a question whether it is appropriate for the NJHA to allocate its funds to a project which only can benefit a very select and small portion of its membership—the 20 to 25 hospitals which may enter into an HMO relationship with the AFL-CIO.

I have been an employee of MSNJ for 18 years. During that time, MSNJ always has dealt openly and fairly with the NJHA. Our candid approach to matters of mutual concern has not been reciprocated in the instance of this HMO project. As a matter of fact, representatives of the Association, when approached at open meetings with MSNJ representatives, have been—at best—evasive. Patients, hospitals, and doctors have a large stake in the current and future health care delivery system in New Jersey. The Hospital Association's effort to affect a major change without an open discussion with all hospitals, their medical staffs, and MSNJ is a mistake. Hospital medical staffs and governing boards should demand a full and honest disclosure from the NJHA on its HMO joint venture with the AFL-CIO. If that disclosure is not forthcoming, an absolute barrier to meaningful relations between the NJHA and MSNJ will have been erected at a time when organizational cooperation is a critical necessity. There is enough grief and anguish being directed at doctors and hospitals. None of us need, nor should we stimulate, divisiveness in the currently difficult and chaotic tenor in which we are forced to function.

We should all remember what happened to the participants in the original "unholy alliance."

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*The material for this column is coedited by Arthur Krosnick, M.D., Editor, *NEW JERSEY MEDICINE*, MSNJ; Vincent A. Maressa, J.D., Executive Director, MSNJ; and James E. George, M.D., J.D., Director of Professional Liability, MSNJ.

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Low incidence of side effects
CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

*Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

References:

1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

Reduces angina attack frequency*
42% to 46% decrease reported in multicenter study.[1]

Increases exercise tolerance*
In Bruce exercise test,[2] control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes ($P<.005$).

CARDIZEM®
(diltiazem HCl)

THE BALANCED CALCIUM CHANNEL BLOCKER

Please see full prescribing information on following page.

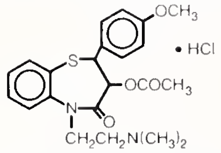
2/84

PROFESSIONAL USE INFORMATION

cardizem®
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy)-5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+)-cis-. The chemical structure is:



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect; cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given; a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS).

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral/LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy**—CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other.

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Another patient benefit product from

PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MISSOURI 64137

STATE OF THE ART:

IDENTIFYING THE ALCOHOLIC PATIENT IN YOUR PRACTICE

Many skilled practitioners lack the knowledge or are embarrassed when they confront alcoholism in their own patients. They are unaware of the prevalence of the clinical manifestations of the primary disease of alcoholism and how to diagnose it accurately. Such a diagnosis can and should be made long before the sequelae appear. Some physicians seem afraid to offend their patients by asking questions that might imply the suspicion of a problem with alcohol.

If we can accept abuse of alcohol and/or drugs as legitimate diseases, there should be no timidity in including them in a historical review.

National statistics[1] tell us many things:

1. Between 10 and 13 percent of the adult population in this country is alcoholic;
2. More than 50 percent of motor vehicle fatalities and injuries are associated with alcohol abuse;
3. Among general hospital patients, 25 to 30 percent have alcohol-related illnesses or injuries; in inner-city hospitals this figure approaches 40 percent;

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Physicians are confronted by alcoholic patients in their practice. The widespread prevalence of this disease and its impact mandate awareness of the incidence and a diagnostic game plan to identify the alcoholic patient.

4. About 20 percent of visits to the emergency room or to the primary care physician's office involve patients with a significant alcohol problem;
5. One-third of families have problems secondary to alcohol;
6. Thirty percent of the drinking population consumes 80 percent of the alcohol consumed; and
7. Thirteen percent of the population (the alcoholics and smokers) use 50 percent of the health care benefits.

All of these statistics indicate that alcoholism is one of the most common diseases you will confront in your practice.

In the easiest scenario, the patient comes into the office and says, "Doctor, I think I have a drinking problem."

The alcoholic's denial is so strong, that for an individual to appear in your office with such a complaint almost is diagnostic in itself. While there may be an occasional exception to this rule, my personal experience is that it is very rare—that individual is in your office because he is rightfully concerned, his spouse or his family is justly concerned, or his boss is "on his back." A physician error at this point can do serious harm.

What do you think your present success rate is in terms of the correct primary diagnosis of this disease among your patients? The following review of the many apparent indicators may improve this particular diagnostic skill:

The physician needs personal guidelines to measure the patient's alcohol use and to assess normal or abnormal nature. A very popular joke among alcoholism counselors says: "An alcoholic is someone who drinks more than his doctor." This implies that the physician's yardstick often is his own behavior (however normal or abnormal that may be): "I drink that much and I don't think I have a problem!"

Don't fall into this trap! Be objective. If the physician is too ready to downplay and belittle the patient's concern, recovery may be delayed for a significant period of time. An incorrect professional opinion is a credential that the patient will use to silence a heartsick spouse, a concerned employer, or a nagging conscience. The astute clinician will be sure of his ground before dismissing the possibility that the patient justifiably may be concerned.

Occasionally the physician may be in a bind when a patient's spouse calls in advance of the patient's appointment to ask the doctor to do something about the spouse's drinking problem while at the same time refusing to give permission to mention that call or the source of prior knowledge. If the patient, caught up in denial, refuses to admit to any problem with alcohol and skillfully dodges questions, the doctor may be unable to proceed in that area without other solid evidence. Some of the information that follows may provide the necessary tools to deal with that situation.

Physicians always must bear in mind that there are no pathognomonic physical signs or symptoms of early alcoholism. The earliest signs are behavioral and are elicited only by a careful history. By the time one is able to detect physical signs, he no longer is dealing with early alcoholism. In the early stage, the patient is more liable to seek treatment because of the consequences of his alcohol-induced behavior: traumatic injuries (at home, on the job, or on the highway); burns; unexplained bruises; gastrointestinal problems such as chronic indigestion, chronic diarrhea, nocturnal or morning vomiting, a peptic ulcer; and a stress-related condition: hypertension, sleep disturbances, or anxiety attacks.

CLINICAL HISTORY

In taking the clinical history of these patients, special attention must be given to certain areas:

The Family History. A family history will reveal the presence of alcoholism, drug abuse, or teetotalism. The physician must pursue this beyond the immediate family to the parents, the parents' siblings, and their ancestors. Be suspicious concerning a father dead at an early age of an accidental injury or a parent who abandoned the family. If the patient is an alcoholic, a positive family history almost always will surface if you search hard enough.

The History of the Patient's Alcohol Use. Nowhere in medicine does the physician have to be more delicate and persistent than in extracting a reliable drinking history. Ask about specific amounts consumed, the frequency of alcohol use, and the usual pattern of drinking behavior.

The alcoholic has been defined as: "Someone who drinks between drinks." This is an accurate description. The astute clinician needs to ask the patient: Have you ever felt the need to cut down? Have you ever gone on the wagon? Have you ever decided to switch beverages? Have you ever continued to drink when it was not in your own best interests? Does alcohol do something for you? Does alcohol make you feel different? Can you remember your very first drink? Do you need a drink to relax, to sleep, or to be sociable? Do you usually get drunk or can you hold more than anyone else in your crowd? Do you ever lose control of your drinking? Have you ever had a blackout? and Have you ever had a seizure after you stopped drinking?

Much of this drinking history readily can be unearthed by the use of a special questionnaire. A popular one is MAST—Michigan Alcoholism Screening Test (Figure), which contains 25 alcohol-related questions weighed for relevance. With a score of more than 5 points, the patient probably is alcoholic. With a score of more than 9 points, the patient usually is alcoholic. The average score of patients in an alcohol rehabilitation program is 24 points. The physician often can go through this test with the patient, leading the patient to draw his own conclusion and to make his own diagnosis.

Another simple test is called the C-A-G-E Test.[2] Only 4 questions are used here: Have you ever felt the need to Cut down? Do other people's complaints about your drinking Annoy you? Do you ever feel Guilty about your drinking? Do you sometimes need an Eye opener? The authors of this test suggest that two positive responses indicate a 90 percent risk of alcoholism.

ALCOHOL-RELATED PROBLEMS

Family. Marital problems are common. The spouse usually is concerned or angry. The alcoholic's children often have behavior and adjustment problems. A history of child abuse may be present. An increased amount of psychosomatic disorders in the family is not unusual.

Job. Is the patient overqualified for the job he holds? Have there been frequent job changes, career changes, or failure to advance in the company? Multiple geographic relocations, multiple warnings from supervisors, and financial difficulties are all clues.

Societal. Common problems include auto accidents, arrests for driving while intoxicated, fights or brawls in the intoxicated state, arrests as a disorderly person, and the history of being banned from local taverns.

Attitude. The patient's attitude about the doctor's questions is very important. Does he belittle the problem? Does his reaction represent an inappropriate response—all a big joke? Is he angry regarding questioning? Does he defend his right to drink or attempt to change the subject? All of these should be an indication that a problem exists.

The following is a practical definition of alcoholism: "Alcoholism is repetitive, excessive drinking, usually beyond the control of the individual, continuing even when it no longer is in the individual's best interest, and productive of serious problems in one of the major areas of life."

The Physical Examination. There often are subtle clues that you can observe and which may indicate a problem with alcohol abuse.

The odor of alcohol on the patient's breath, especially early in the day, should arouse concern. The effects of alcohol withdrawal on the alcoholic (even as early as 12 to 24 hours after the last drink) may cause signs of neuromuscular irritability to be present. The patient may be unusually irritable, obviously tense, restless, and anxious.

Look for a fine tremor or the presence of vasomotor signs such as excessive perspiration with a moist upper lip, wet hair, or an exaggerated puddle sign on the paper on the examining table. Remember that not all vasomotor signs in middle-aged female patients are due to menopause. Don't be afraid to suspect alcohol abuse in the wife of a colleague or even the wife of the parson—no group is immune from this disease.

The physical sequelae of alcohol-related trauma such as multiple bruises or the scars of old injuries may be an additional clue. It is only when the disease is moderately advanced that we begin to see changes in the physical examination that are valid reasons to suspect alcohol abuse as their cause. Included here are the following:

Hypertension. Usually moderate in degree, hypertension occasionally may be quite high. This is a common finding which often returns to normal with abstinence from alcohol.

Cardiac Arrhythmias: Tachycardia and premature ventricular contractions may result from toxic myocardiopathy or electrolyte disturbances due to prolonged vomiting and/or diarrhea. Arrhythmia may be a part of the withdrawal response.

Enlargement of the Liver: Hepatomegaly is always a cause for concern. It may be due to one of the three forms of alcoholic liver disease: 1) Fatty infiltration when the liver is moderately enlarged, smooth, and nontender; 2) Alcoholic hepatitis when the liver is tender. The patient may complain of right upper quadrant abdominal pain. This condition generally is associated with jaundice and a low-grade temperature, and can mimic an acute gallbladder attack. Correct diagnosis is essential because inappropriate surgery in these cases has a 5 to 10 percent operative mortality; 3) Cirrhosis of the liver where the liver enlargement varies widely. The liver surface often is nodular and the liver may be tender especially in the later stages; jaundice is a later manifestation.

While positive differentiation of these forms of alcoholic liver disease require liver biopsy, the physician usually can be quite precise in this differential diagnosis.

Burns on the Fingers and Chest: These usually are due to the patient's tendency to fall asleep while smoking. They are highly suggestive of substance abuse (alcohol or drugs).

Telangiectatic Vessels: These occur with regularity especially around the face.

Spider Angiomata, Gynecomastia, and Testicular Atrophy: These are nonspecific but often are seen in chronic alcoholism. They are felt to be caused by impaired estrogen metabolism in the liver and may occur individually or in any combination.

Peripheral Neuropathy: Neuropathy always is a reason to suspect the presence of alcoholism. It is due, in part, to the nutritional deficiencies that result when the appetite is satisfied by calories derived from alcohol.

Ascites: Although it is usually a later sign and associated with other signs of liver damage, ascites may appear early.

Peripheral Edema: When edema is not due to the more common causes, it also may be an early sign.

The Alcoholic Facies: Characterized by puffiness, telangiectasis, and bilateral parotid swelling, the alcoholic facies is not hard to recognize. It usually is associated with advanced disease. While physical examination may not demonstrate any characteristic changes in the following instances, the presence of any of these diagnoses should alert the physician to a possible alcoholic basis: peptic ulcers; pancreatitis; gastrointestinal bleeding; a Mallory-Weiss tear at the esophagogastric junction; chronic diarrhea; osteonecrosis of the femoral head; seizure disorders in the adult; and organic brain damage.

Finally, the presence of the manifestations of the alcohol withdrawal syndrome, tremulousness, hallucinosis, and delirium tremens generally are easy to recognize and are readily accepted diagnostic makers.

LABORATORY DATA

Considerable attention has been given to the laboratory as the possible source of objective criteria to confirm the clinical suspicion of the diagnosis of alcoholism before significant changes occur in the physical examination. There are a few tests that appear to be valuable at that stage.

A report from Wayne Burton and Lee Gladstone at Northwestern University in Chicago[3] suggests that a battery of simple tests can provide an effective screening mechanism in the physician's office. They call their battery the M.I.L.T. test and it includes: M: Mean corpuscular volume of the red blood cells (MCV); I: Serum iron level; L: Liver function tests; and T: Serum triglycerides.

MCV is frequently elevated in chronic alcoholics. Macrocytosis may persist for weeks or months after abstinence from alcohol and the restoration of good nutrition. It takes a prolonged period to develop and is an index of chronicity.

Serum iron usually is increased, in spite of the macrocytosis, unless there has been significant blood loss or a significant dietary deficiency in iron. Macrocytosis without anemia is a strong indicator of alcoholism.

The liver function studies that are most likely to be abnormal include SGOT, LDH, and serum bilirubin. Elevation of these levels is common.

Triglyceride levels have been known to be increased by the ingestion of alcohol—even in nonalcoholic patients. Clinicians should advise their patients to abstain from alcohol for at least three days before testing for these levels.

Elevation of serum iron, liver function tests, and serum triglyceride level in the presence of macrocytosis strongly suggests chronic alcohol abuse. The reversal of these levels with abstinence is confirmatory evidence.

Wilis Maddrey, Professor of Medicine at Thomas Jefferson University Medical School,[4] wrote an excellent review of alcoholic liver disease making two special points:

1. Gamma Glutamyl Transpeptidase (GGTP) probably is the most sensitive of the liver function tests for indicating recent excessive consumption of alcohol. Small to moderate amounts of alcohol do not cause elevation of this enzyme.

2. Liver function tests almost are useless in attempting to classify the type and severity of alcoholic liver disease. Liver biopsy is the only reliable tool. The liver function tests may be normal in patients who are about to die from cirrhosis.

One other laboratory test, the blood alcohol concentration (BAC), has diagnostic significance under certain circumstances. When this level is significantly elevated (e.g. more than 0.15 mg/dl) and the patient shows no signs or symptoms of acute intoxication, the patient has demonstrated tolerance for the effects of alcohol, which in turn suggests addiction to alcohol.

CONCLUSION

The physician needs a set of objective criteria on which to base the diagnosis of alcoholism. A group of physicians at the Mayo Clinic[5] suggest that the following are most useful for the primary physician:

1) Physiological dependence on alcohol is manifested by a withdrawal response (the abstinence syndrome) when alcohol use is discontinued.

2) Evidence of tolerance is the ability to tolerate significant increases in the blood alcohol concentration without apparent effects.

3) A major alcohol-associated illness appears in a person who drinks regularly.

4) Continued alcohol consumption occurs against medical advice or when it no longer is in the individual's best interest to do so, one can recognize alcohol abuse.

5) The patient's subjective complaint occurs when "loss of control" over his drinking is expressed by the patient.

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MICHIGAN ALCOHOLISM SCREENING TEST (MAST)

Points for "Yes"			*YES*	*NO*
	0.	Do you enjoy a drink now and then?	___	___
(2)*	1.	Do you feel you are a normal drinker? (By normal we mean you drink less than or as much as most other people.)	___	___
(2)	2.	Have you ever awakened the morning after some drinking the night before and found that you could not remember a part of the evening?	___	___
(1)	3.	Does your wife, husband, a parent, or other near relative every worry or complain about your drinking?	___	___
(2)*	4.	Can you stop drinking without a struggle after one or two drinks?	___	___
(1)	5.	Do you ever feel guilty about your drinking?	___	___
(2)*	6.	Do friends or relatives think you are a normal drinker?	___	___
(2)*	7.	Are you able to stop drinking when you want to?	___	___
(5)	8.	Have you ever attended a meeting of Alcoholics Anonymous (AA)?	___	___
(1)	9.	Have you gotten into physical fights when drinking?	___	___
(2)	10.	Has your drinking ever created problems between you and your wife, husband, a parent, or other relative?	___	___
(2)	11.	Has your wife, husband (or other family members) ever gone to anyone for help about your drinking?	___	___
(2)	12.	Have you ever lost friends because of your drinking?	___	___
(2)	13.	Have you ever gotten into trouble at work or school because of drinking?	___	___
(2)	14.	Have you ever lost a job because of drinking?	___	___
(2)	15.	Have you ever neglected your obligations, your family, or your work for two or more days in a row because you were drinking?	___	___
(1)	16.	Do you drink before noon fairly often?	___	___
(2)	17.	Have you ever been told you have liver trouble? Cirrhosis?	___	___
(2)†	18.	After heavy drinking have you ever had delirium tremens (DT's) or severe shaking, or heard voices or seen things that really weren't there?	___	___
(5)	19.	Have you ever gone to anyone for help about your drinking?	___	___
(5)	20.	Have you ever been in a hospital because of drinking?	___	___
(2)	21.	Have you ever been a patient in a psychiatric hospital or on a psychiatric ward of a general hospital where drinking was part of the problem that resulted in hospitalization?	___	___
(2)	22.	Have you ever been seen at a psychiatric or mental health clinic or gone to any doctor, social worker, or clergyman for help with any emotional problem, where drinking was part of the problem?	___	___
(2)‡	23.	Have you ever been arrested for drunk driving, driving while intoxicated, or driving under the influence of alcoholic beverages? (If YES, how many times?)	___	___
(2)‡	24.	Have you ever been arrested, or taken into custody, even for a few hours, because of other drunk behavior? (If YES, how many times?)	___	___

*Points are for "no" answer.
†5 points for delirium tremens.
‡2 points for each arrest.

SCORING SYSTEM: In general, five points or more would place the subject in an "alcoholic" category. Four points would be suggestive of alcoholism, and three points or less would indicate the subject was not alcoholic.

Programs using this scoring system find it very sensitive at the 5-point level, and it tends to find more people alcoholic than anticipated. However, it is a screening test and should thus be sensitive at its lower levels.

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HMO FACTS OF LIFE

By now, you've undoubtedly heard about HMOs, IPAs, et al. They're complicated, confusing and causing physicians considerable concern in terms of their present and future practices.

The following clearly defines what each is. After reviewing these various types of pre-paid health care, we believe you will have the basic information necessary to choose the best for you.

HEALTH MAINTENANCE ORGANIZATIONS (HMOs)

CENTER OR CLINIC

- Central facility or facilities
- Staff physicians
- Patient selects a primary care physician (Internist, GP, FP, and Pediatrician) from the staff
- May frequently have physician turnover resulting in short term doctor-patient relationship and/or the need to re-select primary physician

DIRECT PHYSICIAN CONTRACTING

- No central facility
- Private practicing physicians
- Patient selects a primary care physician (Internist, GP, FP, Pediatrician)
- Obstetrician-Gynecologist is not a primary, but a specialist. The patient is seen by the Obstetrician-Gynecologist only by referral from the primary care physician.
- Primary physicians are capitated. That means they are paid a fixed amount annually to treat a specific patient whether they see that patient once—or a hundred times.
- No group voice because there is no physician group
- Specialists are reimbursed a percentage of their fee
- Patients can see physicians when they want

IPA CORPORATIONS

- No central facility
- Private practicing physicians
- Patient selects 1 or 2 primary physicians: Female and male adults select an Internist, GP or FP; females additionally select an Obstetrician-Gynecologist; parents select a Pediatrician, GP or FP for their child(ren).

  Obstetrician-gynecologists are primary care physicians, and can be seen directly by the member for both routine and specialized care with no referral necessary.
- A corporate body which is organized by physicians. The IPA then contracts with an HMO to provide services to the HMO members. *The physician does not join the HMO, but simply participates.*
- The IPA is the physicians' voice to the HMO which represents the physician re: fee reimbursement, quality of care, peer review, etc.
- Traditional fee-for-service payment
- Patients can see physicians when needed. Each visit is paid for by the HMO on a fee-for-service basis

EXAMPLES

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A NEW METHOD FOR MEASURING BODY TEMPERATURE

JOEL R.L. EHRENKRANZ, M.D., FLEMINGTON*

Studies in subjects ages 2 to 83 show that the temperature of freshly voided urine accurately detects changes in body temperature occurring with ovulation and fever. Use of a disposable, colorimetric urinary thermometer makes measuring core body temperature simple, precise, and convenient.

Urine, as a filtrate of blood, is in equilibrium with core body temperature, and the temperature of freshly voided urine is said to represent the most accurate means for measuring body temperature.[1] Prior studies of military, geriatric, inpatient, and normal volunteer populations have demonstrated parallelism between urinary temperature and oral or rectal temperature.[2-5] This paper describes the clinical validation of a newly developed, single-use, disposable device for taking the temperature of freshly voided urine.

METHODS AND MATERIALS

Clinical thermometers were obtained from the following: urinary thermometers from Franklin Diagnostics, Inc., Morristown, New Jersey; electronic thermometers (IVAC Model 321) from IVAC Corporation, San Diego, California; and mercury-in-glass oral, rectal, and basal body thermometers from Becton-Dickinson Co., Rochelle Park, New Jersey.

Urinary thermometers consisted of a colorimetric temperature-sensing element affixed to a thermodynamically designed collecting cup (Figure 1). The temperature-sensing element functions as a peak detector, registering maximum urinary temperature within 45 seconds of immersion and maintaining this temperature indefinitely.

Urinary, mercury-in-glass, and electronic thermometers were calibrated in vitro by submersion of each type of temperature-sensing element in water baths of set temperatures and comparing the temperature registered against that obtained by a National Bureau of Standards calibrated thermometer (Figure 2).

The sample consisted of male and female ambulatory inpatients and outpatients (n = 121) and normal individuals (n = 15), aged 2 to 83. The following populations were studied:

1. Ambulatory emergency room patients undergoing temperature measurement as part of routine clinical assessment (Figures 3A and 3B).
2. Untreated patients with signs, symptoms, and urinalysis consistent with urinary tract infection or with bacteriologic evidence of infection (greater than 100,000 colonies of a pathogenic organism per cc of urine) (Figures 4A and 4B).
3. Normal subjects collecting sequential urine samples through a 24-hour cycle for periods of 1 to 30 days (Figure 5).
4. Spontaneously ovulating normal females measuring basal body temperature as part of routine fertility evaluation (Figures 6A and 6B).

The subjects were instructed to void into the urine collector holding it as close as possible to the body.

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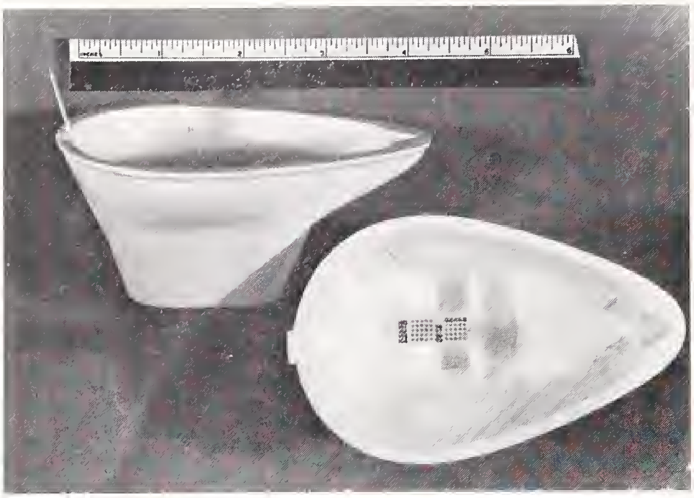


Figure 1—Single-use, disposable urinary thermometers consist of a colorimetric temperature-sensing element positioned within a thermodynamically designed collecting cup.

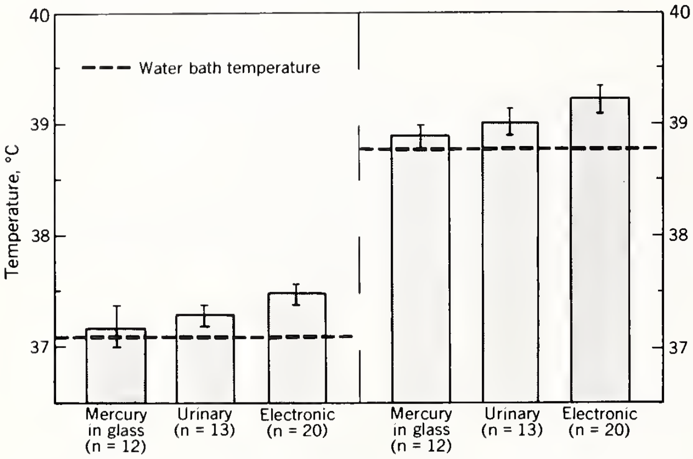


Figure 2—In vitro calibration of mercury-in-glass, urinary, and electronic thermometers. Water temperature of two baths was determined by a National Bureau of Standards calibrated thermometer and is depicted by the dotted line. Mean ± standard deviation of the water temperature as measured by mercury-in-glass, urinary, and electronic thermometers is displayed on the Y axis.

Individuals with sample volumes less than 20 cc (8 percent of subjects) were excluded from the study. After allowing the sample to stand for at least one minute, the temperature-sensing element was removed and read; remaining urine was retained for urinalysis as needed. Oral and rectal temperatures were measured using electronic thermometers according to instrument instructions. Temperatures using mercury-in-glass thermometers were measured with a dwell time of five minutes. All temperatures from an individual subject were measured within 15 minutes of one another.

Statistical analysis was performed with use of the least squares method to calculate the coefficient of correlation.[6]

RESULTS

In vitro testing demonstrated that urinary thermometers were as precise as mercury-in-glass thermometers and more accurate than electronic instruments. In both normal and patient subjects, urinary temperature correlated significantly ($P < .001$) with rectal (r = .92) and oral (r = .92) temperature. Mean urinary temperature was 0.2°C greater than oral and 0.3°C less than rectal temperature. This linear relationship was not disrupted by the presence of urinary tract inflammation ($P < .01$).

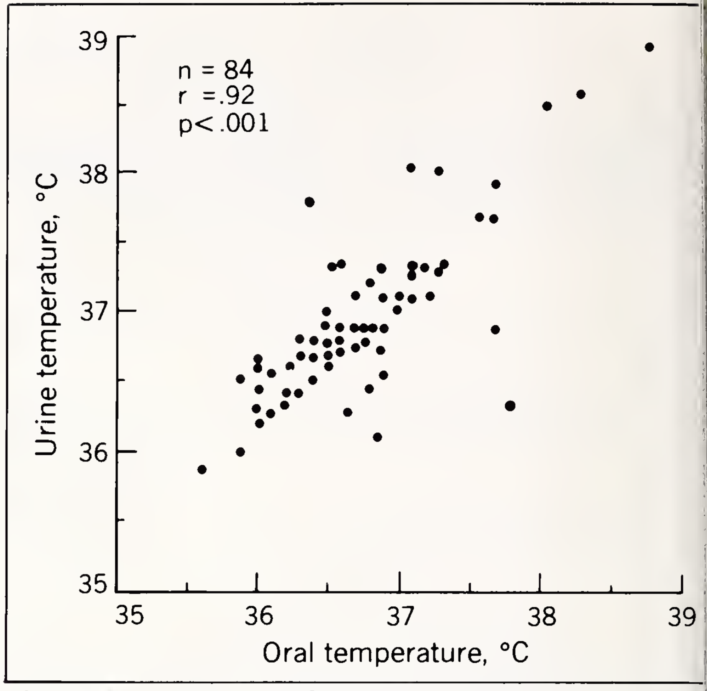


Figure 3A—Comparison of oral versus urinary temperatures. Temperatures for each subject were measured within 15 minutes of one another.

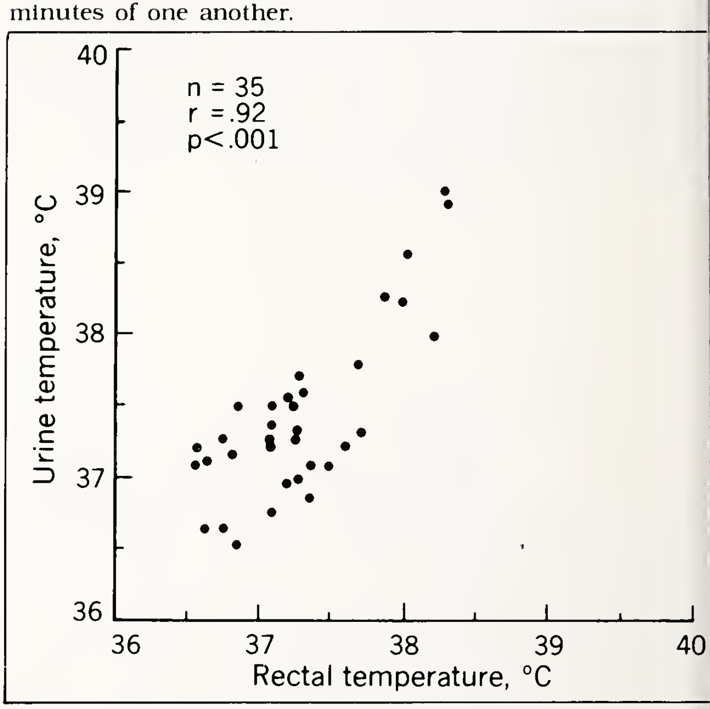
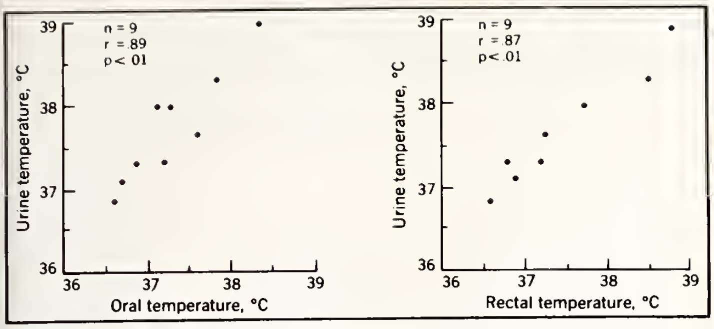


Figure 3B—Comparison of rectal versus urinary temperatures. Temperatures for each subject were measured within 15 minutes of one another.

Additional studies demonstrated a circadian rhythm to urinary temperature in subjects as young as two years of age. In spontaneously ovulating females, parallel and simultaneous rises in urinary and oral temperature occurred at the time of ovulation and persisted through the luteal phase of the menstrual cycle.

DISCUSSION

Although the use of urinary temperature as a measure of deep body temperature has been known for over 50 years,[7] no appropriate instrument for measurement was developed until now. The results described demonstrate the clinical accuracy of a new, single-use, disposable urinary thermometer to measure core body temperature. The correspondence between simultaneous urinary temperature and oral or rectal



Figures 4A and 4B—Urinary temperature in urinary tract infection. Urine, rectal, and oral temperatures were measured within 15 minutes of one another in nine patients with signs, symptoms, urinalysis, or bacteriologic evidence of urinary tract infection.

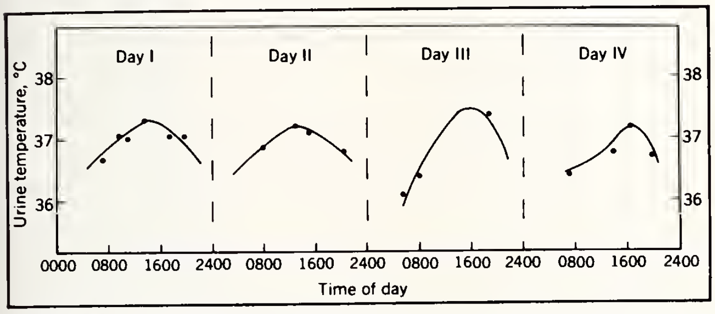


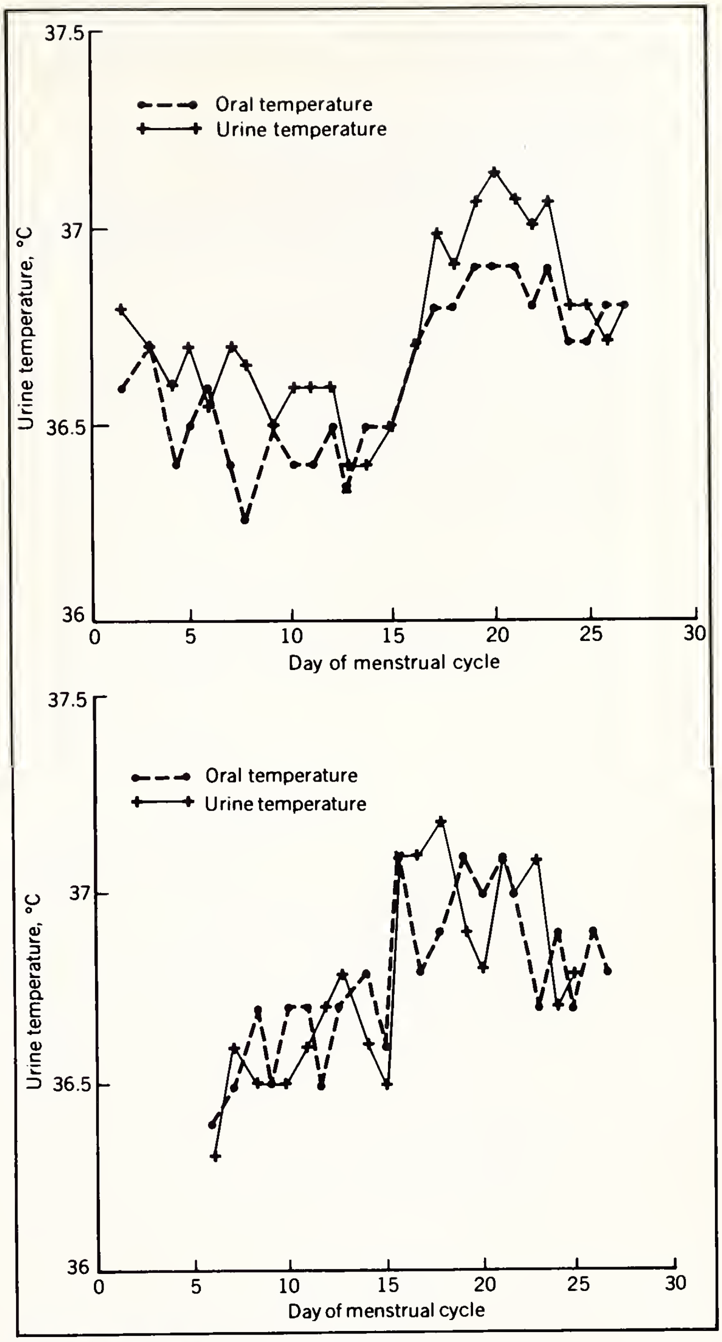
Figure 5—Circadian rhythm of urinary temperature in a two-year-old male. Random urine samples were collected throughout the day over four consecutive days in a normal two-year-old male. Time of day (in military time) is depicted on the X axis; urinary temperature on the Y axis. Characteristic late afternoon peak in temperature is apparent.

temperature is constant and is not disrupted by local inflammation. Urinary temperature reflects alterations in core body temperature due to circadian rhythms and ovulation.

Oral and rectal thermometers have several drawbacks. Oral temperature measurements require meticulous attention to technique because oral temperature varies by as much as 0.9°C based upon placement in the mouth,[8] and is affected by eating, drinking, smoking, talking, and respiratory rate.[9] Rectal temperature, although insulated from ambient conditions, represents the highest regional temperature in the body, exceeding intracardiac temperature by 0.24°C. Its thermal inertia is large, making it less sensitive than oral, blood, fat, or muscle temperature to acute fluctuations in core temperature.[10,11]

There also are several hazards associated with oral and rectal temperature taking. The use of mercury-in-glass thermometers requires insertion of instruments containing toxic amounts of mercury into the body.[12] Additional hazards associated with rectal temperature taking include rectal perforation and the production of bacteremia.[13,14]

The urinary thermometers used in the studies reported here are not subject to any of the risks or problems associated with oral or rectal thermometers. Furthermore, the patient and nursing time required for urinary thermometry is considerably less than that required with mercury-in-glass thermometers. With basal body temperature determination, urinary thermometry requires two hours less per month than methods employing traditional mercury-in-glass basal body thermometers. According to the manufacturer, urinary thermometers will be comparable in cost to mercury-in-glass fever and basal body thermometers.



Figures 6A and 6B—Basal body temperature change throughout the menstrual cycle in two spontaneously ovulating females. Subjects measuring oral basal body temperature as part of routine fertility studies (dotted line) also measured temperature of first morning void (solid line). There is a parallel rise in urinary and oral temperature at the time of ovulation.

The only limitations to the technique of urinary temperature taking would arise from an individual's inability to provide 20 cc or more of freshly voided urine due to altered mental status, incontinence, or oliguria.

In addition to the detection of fever and ovulation, urinary thermometers are particularly well-suited for use in pediatric, emergency room, and geriatric populations.

SUMMARY

Urinary thermometers are not subject to the factors which limit oral temperature's accuracy, are more convenient and significantly less distasteful than rectal temperature measurement, and are safer and noninvasive as compared to either mercury-in-glass or electronic thermometers. Urinary temperature measurement, therefore, should become the method of choice for measuring body temperature in clinical practice.

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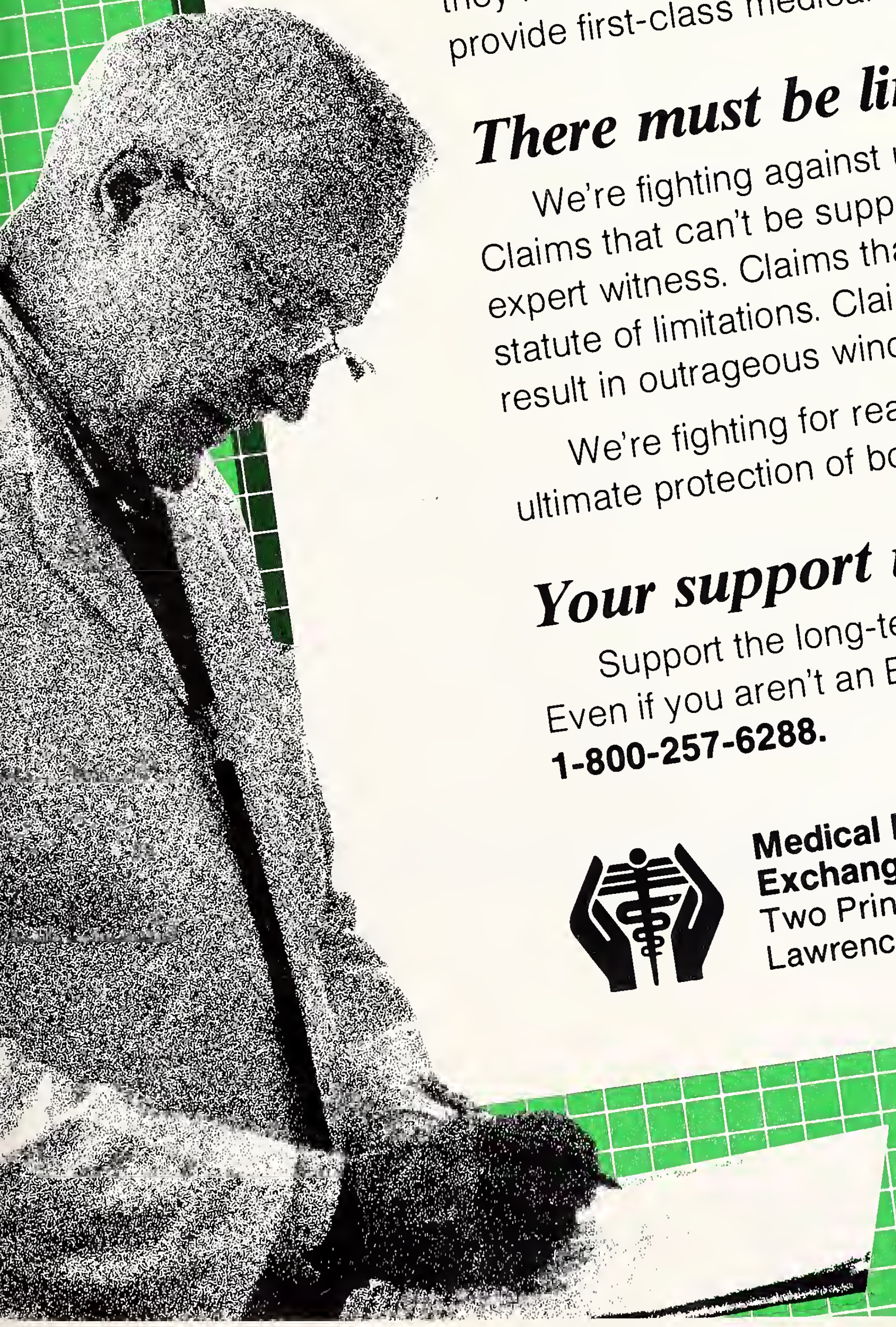

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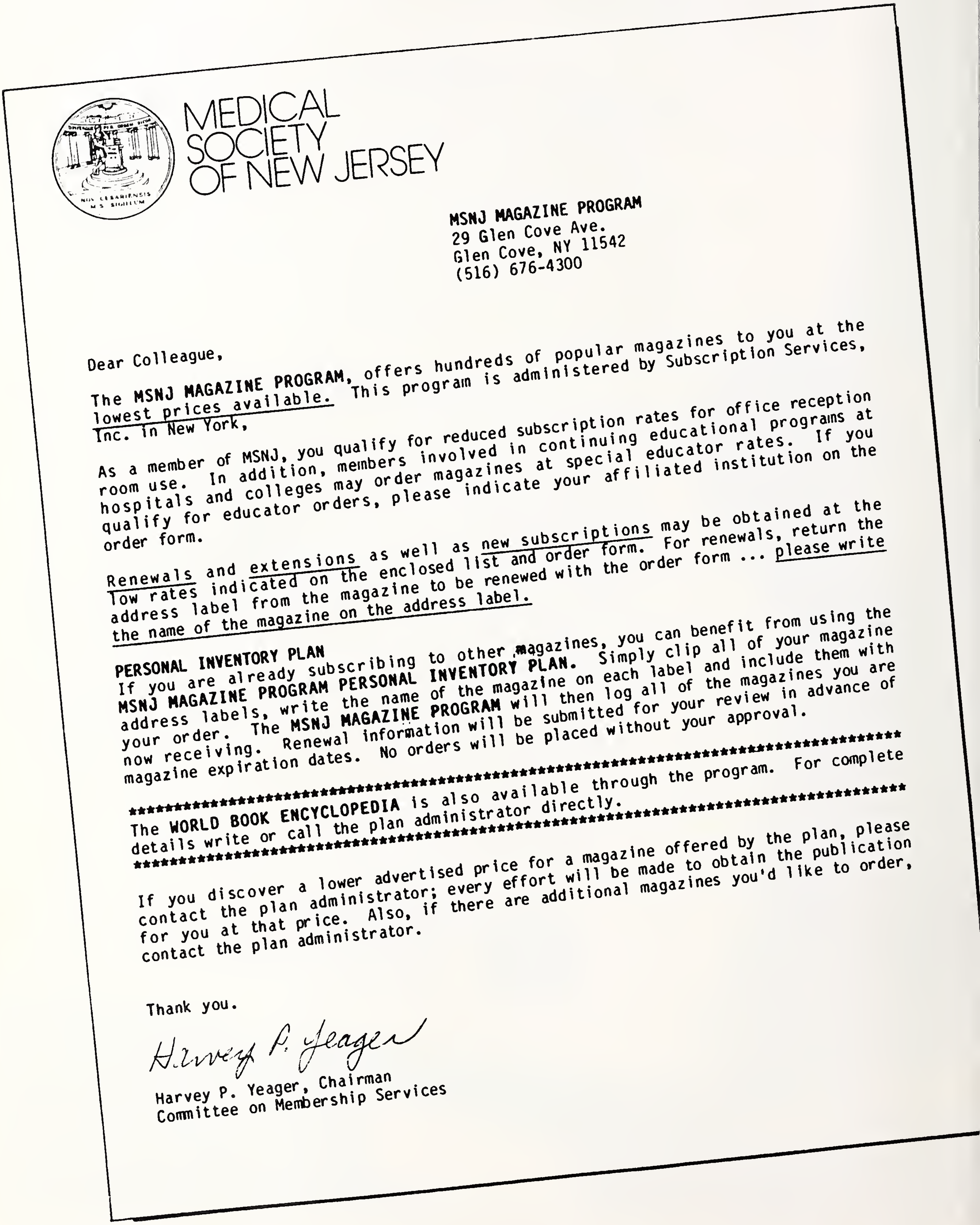

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RADIONUCLIDE SCANS TO DEFINE PATTERNS OF OCCULT MYONECROSIS

JEFFREY L. KAUFMAN, M.D., STEVEN T. DEAK, M.D., WILLIAM ERDMAN, M.D., NEW BRUNSWICK*

After revascularization of the acutely ischemic limb, fasciotomy may be necessary. Despite adequate compartmental release, residual myoischemia and myonecrosis may be present. Combination isotopic scans with technetium pyrophosphate and thallium facilitate the management of this ischemic muscle.

After a brief period of time, usually four to six hours, myonecrosis and myoedema within a fixed fascial compartment become significant features of acute arterial occlusion. Early fasciotomy usually is performed on the basis of clinical suspicion or on the evidence of increased fascial compartment pressure. At fasciotomy, bulging edematous muscle masses usually are found, and their viability is assessed at the bedside. This determination may require a period of observation for return of motor function or operative debridement of superficially necrotic muscle to reveal viable deep muscles.

We cared for two patients with complicated episodes of acute arterial occlusion and myonecrosis, for whom precise clinical assessment of muscle loss was impossible. In this report, we describe the utility of isotopic nuclear medicine scans with thallium-201 and technetium pyrophosphate to determine the pattern and amount of viable muscle remaining after acute arterial occlusion.

CASE REPORT 1

A 19-year old man suffered a motor vehicle accident and initially was treated for a ruptured diaphragm and splenic lacerations. Mediastinal widening was noted on the first postoperative day; angiography demonstrated an aortic injury with free intimal flaps at the level of the ligamentum arteriosum. Repair of the aorta without shunting with a Dacron® graft was performed. Postoperatively, decreased femoral pulses were noted. Six hours later, an angiographic study demonstrated a technical error, a luminal stenosis at the distal graft anastomosis. At re-exploration, the technical error was corrected with restoration of perfusion to the lower extremities after a total ischemic time of 12 hours. Compartment syndromes were recognized early, and bilateral fasciotomies were performed medially and laterally in the legs and thighs. Despite decompression of all compartments, myoglobinuric renal failure occurred. When the patient had recovered sufficiently from anesthesia, he was unable to move his legs and was thought to have both ischemic peripheral neuropathy as well as a central spinal cord injury related to aortic cross clamping. The degree of myoedema became massive; although myonecrosis was certain in the gastrocnemius and soleus groups bilaterally, some deeper muscle groups were questionably viable in both legs. Technetium pyrophosphate and thallium-201 scans were performed (Figure 1). No viable muscle was demonstrated in the right leg; viable deep groups were

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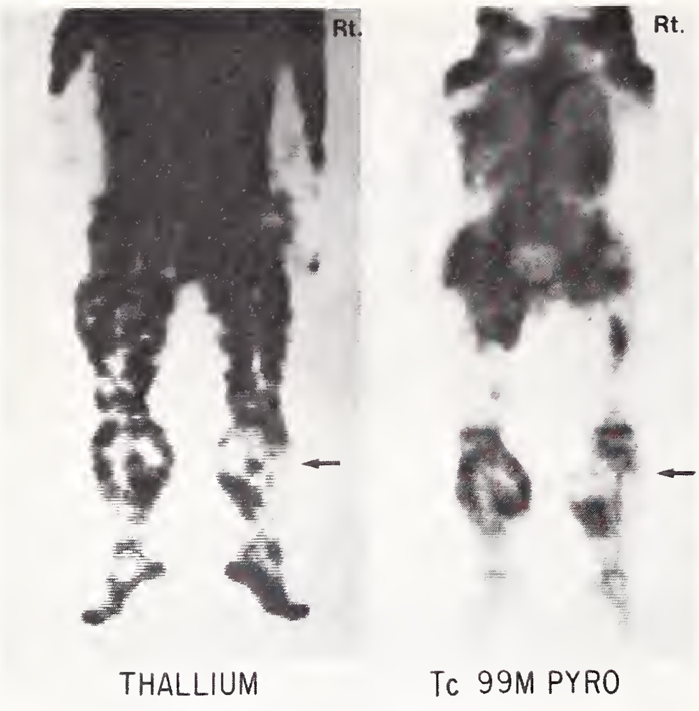

Figure 1—Patient 1. Posterior view technetium and thallium scans. Arrows point at right calf, with massive nonviable muscle (overall absence of thallium and technetium uptake). Left calf, gluteals, and right thigh are perfused (dark shade showing uptake on thallium scan) but injured (dark shade demonstrating abnormal uptake on technetium scan).

seen in the left leg; occult myonecrosis in the gluteals was confirmed. The left leg was debrided conservatively and right below-knee amputation was planned. Before this could be performed, on the 14th postoperative day, the patient suffered sudden respiratory arrest, anoxic encephalopathy, and brain death.

Comment: This patient had mixed neurological and muscle damage. In the early postoperative period, after extensive fasciotomy, it was impossible to determine the prognosis for future motor function. Although there was likely no viable muscle in the right leg on the basis of physical examination, there was hesitancy to perform a definitive above-knee amputation because of the patient's youth and intact arterial circulation. The left leg appeared to have necrosis of the large posterior muscle groups, but enough deep muscle viability to allow for future function. Isotopic scans confirmed these impressions and allowed planning for appropriate debridement and amputation. The scans also disclosed extensive myonecrosis in the proximal muscle groups and gluteals not normally subject to fasciotomy.

CASE REPORT 2

A 58-year old man, who had a history of mild bilateral claudication, was found to have a previously unrecognized abdominal aortic aneurysm at the time of left colectomy for malignant polyps. Postoperatively, decreased perfusion to the right foot was noted, likely due to embolism from intraoperative manipulation of the aneurysm. On angiography, a free-floating popliteal artery thrombus was noted distal to a right superficial femoral artery occlusion. Because of the distal thrombus, as well as the likelihood of atheroembolism, exploration of the femoral and popliteal vessels was performed. There was no embolic material in the common femoral artery or its branches, and the popliteal clot was removed successfully. After this procedure, the foot was nonviable, so re-exploration with placement of a femoral-popliteal bypass graft was performed. The total ischemic time was ten hours. The muscles of the right lower leg became tense, consistent with a compartment syndrome. Limited fasciotomies were performed after eight hours of reperfusion by opening the leg incisions medially and with subcutaneous fasciotomy anterolaterally; the foot perfusion returned to normal. Although myoglobinuria was demonstrated, no alteration of renal function occurred. Technetium pyrophosphate and thallium-201 scans were performed (Figure 2). The deep calf muscle groups were viable, but there was evidence for ischemic damage to the gastrocnemius and soleus, as well as inadequate release of anterior compartment with myonecrosis of the foot extensors. On the basis of the scans, the fasciotomy of the anterior compartment was widened, and the anterior compartment muscles were debrided completely.

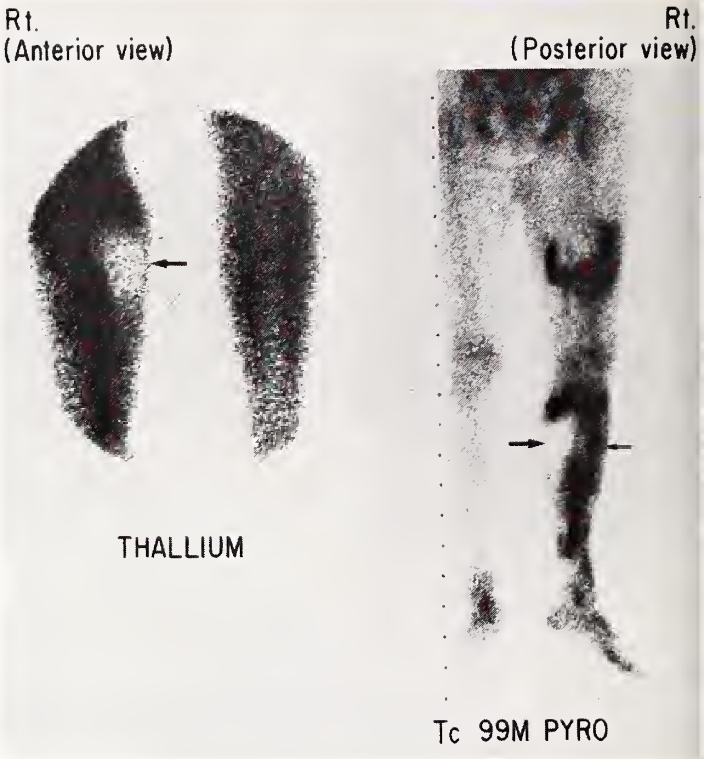

Figure 2—Patient 2. Anterior view below-knee thallium scan on the left, technetium scan of lower half of body on the right. Matched nonperfused nonviable muscle defects (large arrows) in the gastrocnemius-soleus group (absent uptake in both scans). Ischemic perfused anterior compartment group (small arrow), normal uptake on thallium scan, abnormal increased uptake on technetium scan. Dark area on thigh is from inadvertent placement of a urinary collection bag during the scan.

Comment: This patient had both prolonged right lower extremity ischemia and delayed recognition of a compartment syndrome. These problems were complicated by the use of a limited subcutaneous anterior fasciotomy. Isotopic scans demonstrated the need for extending the fasciotomies and delineated a pattern of injured but viable gastrocnemius-soleus muscles sufficient to allow long-term recovery of motor function.

DISCUSSION

After acute arterial occlusion, a fasciotomy frequently is performed at the time of revascularization if the period of ischemia has been prolonged, usually greater than four to six hours. If the fasciotomy is not performed at the time of revascularization, it is the duty of all who care for the patient to recognize the signs of a developing compartment syndrome and to perform an early fasciotomy at the bedside. If the fasciotomy is timely, the released pouting muscle may be edematous but obviously viable. Although the patient may be unable to move his foot voluntarily, the muscle may respond to touch or to a nerve stimulator. If perfusion of the foot is improved after fasciotomy, and distal sensation is observed, then the prognosis for long-term function is good.

In cases of delayed treatment of a compartment syndrome, frank necrosis of whole muscle groups or interspersion of both viable and nonviable muscle fascicles may occur. As in the case reports, this complicated picture of myonecrosis may be associated with neurological damage.

What is the best method for treating this larger mass of necrotic tissue? In most instances, removal has relied on a combination of surgical debridement and host resorption, a method complicated by prolonged exposure of the host to myoglobinemia and possible associated myoglobinuric renal failure. Sepsis may occur in the large volume of necrotic tissue. Limb deformity and joint contraction may prevent resumption of normal gait. Surgical debridement may entail a large blood loss due to incision of a well-perfused vascular bed within the dead muscle mass. For these reasons, early delineation of the pattern of irremedial muscle damage is advantageous in cases where the ischemic insult is complex. In the cases noted above, isotopic scans provided this information.

The physiological basis for the use of technetium pyrophosphate and thallium-201 scans has been well documented. Technetium pyrophosphate has been used to demonstrate myocardial necrosis, and an incidental finding has been striated muscle necrosis as may occur in defibrillator burns.[1] The mechanism of action involves the binding of a radio-label complex to damaged myochondria. Uptake is proportional to the extent of myonecrosis.

Technetium pyrophosphate also has been useful for assessment of extensive and occult myonecrosis, associated with electrical burns, and is analogous to the lesions found in our cases.[2-4] Thallium-201 uptake is proportional to muscle perfusion, with a mechanism related to normal membrane transport of the isotope as a potassium analog.[5-6] By combining the technetium pyrophosphate and thallium-201 scans, information is provided about three tissue zones: a) perfused, nonischemic muscle (normal thallium uptake, normal technetium-pyrophosphate uptake); b) perfused ischemic muscle (area of normal thallium uptake with abnormally increased technetium pyrophosphate uptake); and c) nonperfused, nonviable muscle (area of absent thallium uptake matched with absent technetium pyrophosphate uptake, frequently with a border area of increased pyrophosphate uptake consistent with ischemia). These scans are distinctly different from those obtained in isotopic angiography[7] or in washout techniques for the determination of skin or muscle blood flow.[8] The scans are performed with standard imaging equipment, are repeatable, and employ isotopes with a short halflife.

Isotopic scans do take time and are inappropriate in the early assessment of a muscle compartment for planned fasciotomy. The scans are not necessary to assess a postrevascularization, postfasciotomy limb with obviously viable muscle, as is the situation in most patients.

However, in the assessment of the more uncommon complicated cases of mixed ischemic and necrotic muscle, particularly in the later followup after treatment of acute arterial occlusion, isotopic scans are most helpful. They may indicate incomplete fascial release, may delineate occult muscle injury, and may aid in the planning of an appropriate amputation or early debridement of specific muscle groups.

SUMMARY

We have described two patients who suffered acute arterial occlusion with eventual restoration of normal circulation to the legs, but with residual complex patterns of muscle injury or necrosis. Despite treatment with standard fasciotomies, both patients had residual areas of muscle injury or necrosis demonstrated by combined Tc-pyrophosphate and thallium scans but not evident by clinical examination of the postischemic leg. On the basis of the scans, fasciotomies were extended, specific muscle groups were debrided, and appropriate amputations planned. The scans are a useful adjunct in the late assessment of the revascularized extremity with complex muscle injury after acute arterial occlusion.

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CASE REPORT: MULTIFORM ATRIAL TACHYCARDIA

PAUL H. CHEN, M.D., ELIZABETH*

Multiform atrial tachycardia is associated with chronic lung disease. Treatment is directed at underlying respiratory disease. Reported here is a case treated with oral verapamil with reversion to sinus rhythm.

Multiform atrial tachycardia (MAT) usually is found in association with chronic respiratory disease. The pathogenesis of this arrhythmia has not been elucidated and treatment is directed at eliminating the underlying illness. Verapamil has been established to be effective in treating supraventricular tachyarrhythmias, yet its use in MAT has not been recorded. This report concerns a patient with pulmonary malignancy who was found to have persistent MAT. The administration of verapamil resulted in reversion of the arrhythmia to sinus rhythm. The possible mechanism of its action also is discussed.

CASE REPORT

A 75-year-old white man was admitted to St. Elizabeth Hospital in April 1983 because of acute respiratory distress. His pertinent history included a myocardial infarction 15 years ago and chronic obstructive pulmonary disease (COPD) diagnosed 10 years ago. He was hospitalized for COPD with acute exacerbation, and for spontaneous tension pneumothorax which required bullectomy and decortication of the left lung two years ago. He had a cholecystectomy and partial gastrectomy. Backache, loss of appetite, and a 20-pound weight loss were noticed over an eight-week period. More recently, hoarseness also was noticed. Medication included furosemide and aminotriphylline. There was no chest pain, palpitation, fever, chills, or hemoptysis. He smoked two packs of cigarettes per day for over 50 years. One brother died of cancer of the lung.

On admission, his blood pressure was 120/80 mm Hg; pulse rate was irregular between 110 to 130 beats/min; temperature was 100.5°F; and respiratory rate was 28/min. He was cachectic, hoarse, alert, and oriented. Jugular venous pressure was 8 mm Hg without hepatojugular reflux. There was fullness of the left supraclavicular fossa which was firm but nontender. A 2 x 2 cm rock-like supraclavicular node was palpated. Coarse rhonchi and expiratory wheezes were present at both lung bases. The remainder of his physical examination was unremarkable. The initial white blood count was 14,400 cells/mm^3 with 86% polys, 5% bands, and 9% lymphocytes.

Hemoglobin was 11.3 g/dl and serum potassium was 4.2 mEq/L. Arterial blood gases (ABG) on 2 liters of nasal oxygen showed a pH of 7.41, a PCO_2 of 40 mm Hg, and a PO_2 of 69 mm Hg. Chest roentgenogram showed an irregular 6 cm mass lesion in the left upper lobe. Sputum revealed many white blood cells and mixed gram-positive organisms. Electrocardiogram demonstrated a rhythm of multiform atrial tachycardia (MAT) with a variable rate of between 130 and 150 beats/min and frequent ventricular premature depolarizations. The axis was 45° and there were nonspecific ST-T changes (Figure).

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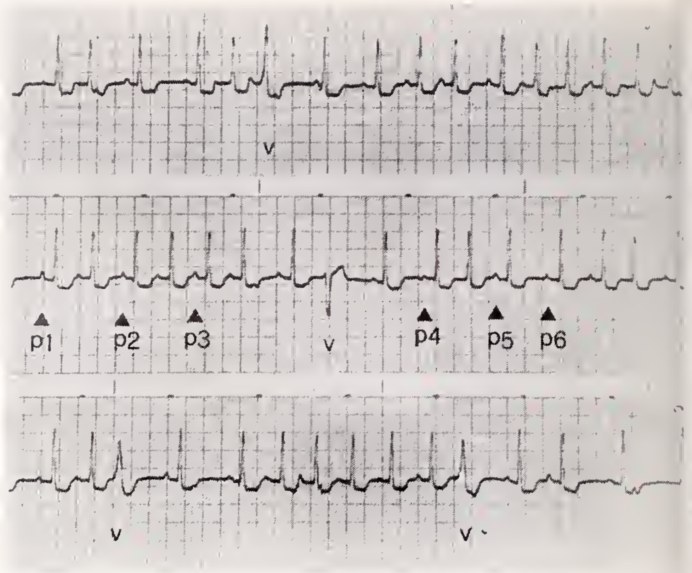
Intravenous aminophylline and antibiotics were initiated and the patient was intubated and treated with a respirator because of deteriorating arterial blood gases (pH 7.29, PCO_2 69 mm Hg, and PO_2 46 mm Hg). Intravenous furosemide was administered for concomitant heart failure. The MAT persisted despite improvement in arterial blood gases (pH 7.45, PCO_2 35 mm Hg, and PO_2 92 mm Hg) and a normal serum potassium. The tachycardia was detrimental to this man with ischemic heart disease, therefore, a test dose of 5 mg of verapamil was given intravenously in an attempt to slow his ventricular responses. This resulted in substantial slowing of his ventricular rate, with no untoward effects on his cardiac status. Two days after initiation of oral verapamil, his rhythm reverted to sinus rhythm with occasional premature atrial contractions. The appearance of isorhythmic AV dissociation five days later was thought to be due to toxic effect of verapamil so the drug was discontinued. His heart rate gradually returned to the pretreatment level despite stable clinical status. Recurrence of MAT three days after cessation of verapamil therapy necessitated its resumption. A serum verapamil level before resuming therapy was 31.8 ng/ml (therapeutic range, 100 to 600 ng/ml). He again reverted to sinus rhythm while on a maintenance dose of verapamil without any ill effects on his cardiac function.

The lymph node biopsy revealed metastatic anaplastic carcinoma of small cell type. He remained in sinus rhythm while on maintenance doses of verapamil, but eventually succumbed to his underlying malignancy and overwhelming sepsis.

DISCUSSION

Multiform atrial tachycardia (MAT) is a poorly understood cardiac arrhythmia and has received very little attention. The pathogenesis of MAT has not been elucidated fully. It generally is found in association with chronic respiratory disease, other debilitating diseases, and congestive heart failure.[1] The diagnostic features of MAT include: an atrial rate of over 100 beats/min, distinct P waves of three or more different configurations, and an isoelectric baseline with varying P-P, P-R, R-R intervals (Figure). By auscultation it may be difficult, if not impossible, to differentiate it from atrial fibrillation because of the irregular R-R interval. The distinct P waves, however, favor the diagnosis of multiform atrial tachycardia. The varying P-R intervals and at times the nonconducted atrial beats separate it from sinus arrhythmia. In case of multiform atrial tachycardia with aberrant conduction, the presence of the distinct P waves preceding each QRS complex differentiates it from ventricular tachycardia or fibrillation.

Treatment should be directed at eliminating the underlying precipitating causes, i.e. severe respiratory insufficiency, infection, or congestive heart failure. It generally is believed that MAT may resolve with improvement in arterial blood gas values but its prognosis is poor, being related intimately to the underlying pulmonary status. In this case, however, MAT persisted despite improvement in arterial blood gas values. Wang et al. reported the use of propranolol in slowing the atrial rate of MAT.[2] Digoxin was used in a subset of patients with MAT where congestive heart failure was thought to precipitate the rhythm disturbance. Treatment of MAT with verapamil has not been reported previously.



Figure—Represents lead II and is a continuous recording. The rhythm is irregular with varying P-P, P-R, and R-R intervals. There are distinct P waves of different morphology as indicated by P1, P2, and P3. There also are ventricular contractions as indicated by V's. Unfortunately, the rhythm strip that demonstrated the isorhythmic AV dissociation was missing. Since the patient was transferred to a nonmonitored bed, rhythm strip which could represent the patient's rhythm before his final demise, could not be obtained. As far as could be ascertained, the patient remained in sinus rhythm while he was still in the intensive care unit one week after the initiation of the second course of verapamil therapy.

Habibzadeh reported a 0.36 percent incidence of MAT among all hospital admissions in a period of 66 months and proposed that far-advanced pulmonary disease, adrenergic stimulation, and age, all play a role in the genesis of MAT.[3] MAT most typically occurs in elderly debilitated patients with severe respiratory impairment. Hypoxia, congestive heart failure, and overwhelming infection are known to cause adrenergic stimulation which may exaggerate atrial automaticity and excitability. On the other hand, age-related degenerative change of the SA node diminishes its automaticity. Multiple atrial automatic foci may be functioning and set the stage for MAT. Chung wrote that the mechanism of MAT is the same as that of unifocal atrial tachycardia except that there are multiple foci in MAT.[4] In their review of the mechanism of supraventricular tachycardia, Josephson et al. point out that there are SA nodal, AV nodal re-entrant tachycardia, and atrial automatic tachycardia.[5] It is conceivable that both automaticity and re-entry play a role in the genesis of MAT.

Verapamil is effective in terminating supraventricular tachycardia (SVT) and in slowing the ventricular rate in atrial fibrillation.[6-9] Its mechanism of action is thought to be due to slowing of the AV conduction which in turn interrupts the re-entrant loop. Furthermore, oral verapamil has been established as effective and safe in preventing recurrence of SVT and in maintaining slow ventricular responses in chronic atrial fibrillation.[10] If re-entry plays any role in the genesis of MAT, it is reasonable to speculate that verapamil may be an effective alternative. Significant slowing of this patient's ventricular response was observed soon after intravenous administration of 5 mg of verapamil. His rhythm reverted to sinus rhythm two days after initiation of oral maintenance verapamil.

CONCLUSION

It is well known that MAT may be transient and may last for seconds to years but generally for less than two weeks.[1] One might argue that the response demonstrated here merely was coincidental, and that spontaneous reversion to sinus rhythm had taken place. The close temporal relationship between administration of verapamil and reversion to sinus rhythm, strongly suggests a cause-effect relationship. This was demonstrated not once but twice in the course of this man's illness. When oral verapamil was discontinued because of the appearance of isorhythmic AV dissociation, MAT recurred within three days. A serum verapamil level drawn then was subtherapeutic (31.8 ng/ml). Arterial blood gases remained stable and serum potassium was maintained over 3.5 mEq/L at all times. The recurrence of MAT could not be attributed to other causes. With resumption of oral verapamil, his arrhythmia reverted to sinus rhythm once again and remained so until his demise. Two additional serum levels drawn on the second and third days after resumption of verapamil therapy were 197.6 and 362.1 ng/ml, respectively. The evidence presented here indicates that verapamil may be useful in treating patients with MAT.

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PEDIATRIC BRIEFS

Pediatric Briefs** are abstracted from the **Newsletter**, a continuing medical education project of the Department of Pediatrics, UMDNJ-New Jersey Medical School and the Children's Hospital of New Jersey.*

Report of the New Jersey Department of Health: How healthy are New Jerseyans? December 1984.
"How healthy are New Jerseyans?" is intended to document the state of wellness/illness of New Jersey's 7,364,823 residents, 48 percent male and 52 percent female. The racial breakdown in 1980 was 83.2 percent white, 12.6 percent black, and 4.2 percent other nonwhite. The document was organized on the basis of life stages and disease or behavioral problems.

Infant Health: Infant death rates declined since 1960. Low birth weights and illegitimate births were still high for nonwhite groups. Infant mortality, 11.7 per 1,000 live births for 1982, was higher than the national average of 11.2 per 1,000 live births due to greater mortality in the neonatal period. New Jersey's white infant mortality rate was 9.6 per 1,000 live births but the nonwhite mortality rate was 19.0 per 1,000 live births.

The neonatal death rate (up to 28 days of age) in 1982 was 8.1 per 1,000 live births. This is higher than the national average and is influenced by the nonwhite neonatal death rate in 1982 of 11.8 per 1,000 live births. The slow rate of decline of the nonwhite neonatal death rate indicates that the national objective of 6.5 neonatal deaths per 1,000 live births will not be met by 1990. Prenatal services subsidized by the New Jersey Department of Health annually provided care to over 9,000 women. The babies of patients served by those subsidized clinics had higher birth weights and lower neonatal death rates than babies of patients from the same communities not served by the clinics.

Child Health: Accidents, congenital anomalies, and malignant neoplasms continued to be significant causes of childhood morbidity and mortality. Accidents remained the number one cause of childhood mortality. Lead poisoning continued to be a problem with over 5 percent of children screened having elevated blood lead levels as compared to 4 percent of children screened nationally. Tuberculosis among children declined. In 1982, 80.4 percent of tuberculosis incidence in children occurred in nonwhite and white-Hispanic populations in New Jersey.

An estimated 400,000 children (22 percent) were raised in single-parent homes and 15 percent of New Jersey's children were in families below poverty levels. In 1980, 53 percent of the 975,000 women over 16 years of age with children under 17 years of age, were in the labor force. Fifty percent of the 90,000 children between the ages of 3 to 4 were enrolled in nursery schools.

In 1982, there were 20,193 cases of child maltreatment of which 35 percent were substantiated and there were 19 deaths. While maltreated children can be found in all income groups, children from low income families are at greater risk probably because of the stresses of poverty.

---

*Editor of the *Newsletter* is Richard H. Rapkin, M.D., Medical Director, Children's Hospital of New Jersey, 15 South Ninth Street, Newark, NJ 07107.

New Jersey school children who were screened between 1982 and 1984 averaged over five decayed, missing, and filled teeth. This is 20 percent above the national average.

During childhood, the common forms of cancer are generally divided into two groups: solid tumors and leukemia/lymphoma. Leukemias are the leading types of cancer in children of both sexes. It is estimated that 470 children in New Jersey were diagnosed as having cancer in 1983. Although the development of multimodal protocols and early treatment have greatly increased the success rate, special problems are created for the child, siblings, and parents by the diagnosis and treatment of cancer.

Adolescents and Young Adults: Accidents were the leading cause of death among the 15 to 24 year olds. Over one-third of the deaths resulted from motor vehicle accidents. Of all adolescent and young adult deaths in 1981, 13 percent were due to homicide and 8 percent were due to suicide. Cancer ranked fourth and caused 7.4 percent of deaths in 1981. Nearly 25 percent of all men and 23 percent of all women between the ages of 20 and 70 were at least 20 percent overweight.

Fertility rate for white girls 10 to 14 years old in 1982 was 0.4 compared to 2.5 for nonwhite girls. Fertility rate for white girls 15 years old in 1982 was 4.9 compared to 30.4 for nonwhite girls. Fertility rate for white girls 16 years old in 1982 was 12.3 compared to 51.5 percent for nonwhite girls. Fertility rates for white girls 17 years old in 1982 was 20.7 compared to 74.6 for nonwhite girls.

Alcohol is the most widely used drug among American youth. A questionnaire administered to 1985 high school students at 29 randomly chosen high schools in New Jersey revealed that 91.2 percent reported having used alcohol, 21.6 percent reported regular use of alcohol (at least 10 occasions within 30 days). A national study concluded that 14.8 percent of students in grades 10 through 12 are heavy drinkers who need professional assistance.

Among household residents over age 12 in New Jersey, 27 percent have used marijuana at least once and 9 percent have used marijuana in the past 30 days. Cocaine was used at least once by 5 percent and 2 percent used cocaine in the last 30 days. Eight percent had used marijuana and alcohol in combination and 2 percent of the population were dependent on drugs and 5 percent admitted having driven a motor vehicle during the past six months while feeling the effects of drugs.

Motor vehicle crashes remained an important cause of death and injury for all age groups, but especially the young. The car occupant death rate per 100,000 population was almost twice as high for infants less than six months of age as for 1-year olds, and three times the rate for children aged 6 to 12. (J. Alexander, M.D.)

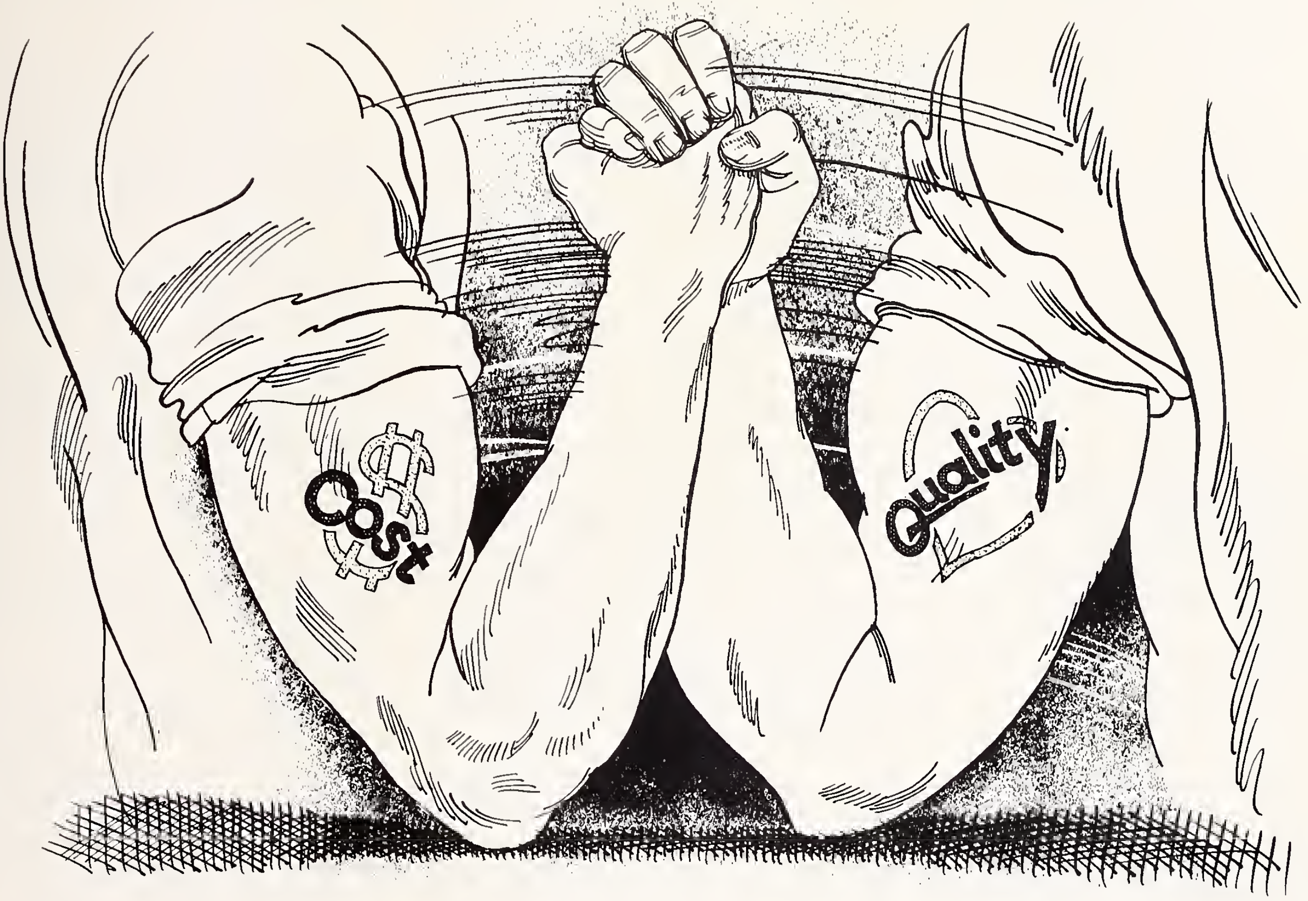
Kramer MS, et al.: Parental fever phobia and its correlates. *Pediatrics* 75:1110, 1985.

Fever is probably beneficial in combating infection and there are no hard data demonstrating fever as harmful (although hyperthermia undoubtedly can be). Middle-class patients' parents were surveyed regarding knowledge about fever. Nearly half considered temperature less that 38°C to be fevers; more than half favored awakening a child for fever treatment. "Undue fear and overly aggressive treatment of fever are epidemic among parents ... considerable effort will be required on the part of pediatricians and other child health workers to re-educate these parents...." One rationale for antipyretic therapy may be to reduce parental anxiety and part of this anxiety "is the message the pediatricians convey to parents."

Comment: I believe that one can educate parents, particularly new parents, to the importance of fever as a defense, its benign significance, and its important suggestion of underlying illness. Equally possible is convincing parents that a major issue in treatment of febrile illness is not fever reduction but rather reducing the discomfort of the child. Reduction of fever is not the same as improving comfort. Intermittent ups and downs of temperature caused by intermittent antipyretic use are very likely more discomforting than persistent fever. We can and will educate effectively our patients' parents when we ourselves believe in the value of such education. It is my responsibility to try to convince my readers of same when the data are consistent and persuasive.

Fleming DW, et al.: Secondary H. influenzae type b (Hib) in day-care facilities. Risk factors and prevention. *JAMA* 254: 509, 1985.

Risk of secondary Hib disease in day care was strongly age related: 2.5 percent in infants under 1 year; 1 percent in 1 to 2 year olds, and nil thereafter. The more time at the day-care center, the more likely the acquisition. Risk of secondary disease in under 2 year olds was almost the same as household contacts. Rifampin substantially reduced this risk. The authors conclude that "the risk of secondary Hib disease for day-care attendees with limited exposure to a primary case appears to be small. However, when the degree of exposure of day care contacts to a child with Hib disease is similar to the degree of exposure that would be seen for household contacts, the risk of secondary disease in day care approaches the risk of secondary disease in households. The child who is less than 2 years old, who attends day care more than 25 hours per week, and who spends more than half his time in the same room as a child with Hib disease is the child who is most likely to develop secondary day care associated disease." Rifampin should be administered to such a child.



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14-058

DOCTORS' NOTEBOOK

Trustees' Minutes; UMDNJ Notes; MSNJ Auxiliary; AMNJ Report; New Members; Physicians Seeking Location in New Jersey

Trustees' Minutes
November 17, 1985

A regular meeting of the Board of Trustees was held on Sunday, November 17, 1985, at the Executive Offices in Lawrenceville. Detailed minutes are on file with the secretary of your county society. A summary of significant actions follows:

Report of Executive Director. . .

(1) MSNJ 1985 Paid Membership . . . Noted total paid membership as of October 31, 1985, was 7,519.

(2) Certificate of Need . . .

(a) Legislation . . . Noted that S-2869 will not come up for a Senate vote before the close of the calendar year.

(b) General Appeal . . . Noted responses to a survey of administrators of NJ hospitals, chairmen of hospital governing boards, and the NJHA are being received.

(c) DRG Appeals . . . Noted a Department of Health proposal to change the composition of the second levels appeals committee to include medical directors of peer review organizations, two physician members of MSNJ, and one physician member of the NJ Association of Osteopathic Physicians and Surgeons.

(3) Preadmission Certification . . . Noted that the Department of Insurance requires all carriers to submit preadmission certification programs to the Department for approval.

(4) State Board of Medical Examiners (SBME) . . .

(a) Pronouncement of Death and Signing of Death Certificates . . . Noted that SBME will pursue matters concerning the definition of "attending physician" which shall be understood to mean the physician who has provided ongoing treatment for the deceased within the three months prior to the date of death.

(b) Assisting Surgeon at Cataract Procedures . . . Requested an opinion from SBME regarding the Health Care Financing Administration policy stating that assisting surgeons were not necessary at uncomplicated surgery procedures.

(5) Medicare/Medicaid Insurance Claim Forms . . . Noted that effective October 30, 1985, physicians are required to purchase Medicare or Medicaid claim forms from either Prudential Insurance Company or the AMA.

(6) Medicare Fee Freeze . . . Noted that the freeze was extended through December 14, 1985.

Council on Legislation . . . Approved the following legislative draft concerning HMOs:

> No health maintenance organization may deny reimbursement for services rendered to any enrollee by any physician licensed to practice in New Jersey if such services were medically necessary and in conformity with accepted standards of practice.
>
> In the event the physician is not a contracting physician or an approved provider by the health maintenance organization, the organization's liability for payment shall be limited to the amount it would have paid if the physician were approved by it.
>
> This act shall take effect immediately.

Committee on Membership Services . . .

(1) Windfall Profits . . . Approved the following recommendation:

> That Resolution #9 (Prevention of Windfall Profits) be referred to the Committee on Health Care Reimbursement Policies.

(2) Legal Services Plan . . . Approved the following recommendation:

> That the Legal Services Plan provided by Kern and Augustine be continued.

(3) Educators Magazine Plan . . . Approved the following recommendation:

> That MSNJ endorse the Educators Magazine Plan to be offered as a membership benefit.

Task Force Coalition with Senior Citizens . . . Requested that an article appear in *NEW JERSEY MEDICINE* calling attention to this Coalition's formation and activities (*J MSNJ* 83:25, 1986).

AMA-Hospital Medical Staff Section-MSNJ . . . Approved the following recommendation and referred it to the joint Executive Committees of MSNJ and NJHA:

> That MSNJ and NJHA jointly study the issue of the integrity of the medical record.

New Business . . .

(1) HMO Contracts in New Jersey . . . Forwarded a letter to the Union County Medical Society advising them of a series of upcoming seminars on HMO issues as well as an analysis of the legal ramifications of HMO contracts.

(2) Unionization . . . Noted a suggestion to advise the membership of the pros and cons of unionization.

(3) Foreign Medical Graduates (FMGs) . . . Noted that NJ Senator Bradley convinced members of the Finance Committee to provide a three year phase-out period for hospitals with less than 50 percent FMGs and an additional two years for hospitals with more than 50 percent FMGs.

Trustees' Minutes
December 15, 1985

A regular meeting of the Board of Trustees was held on Sunday, December 15, 1985, at the Executive Offices in Lawrenceville. Detailed minutes are on file with the secretary of your county society. A summary of significant actions follows:

Report of the President . . .

(1) AMA Delegation Officers . . . Noted that Karl T. Franzoni, M.D., Chairman, and Frank Y. Watson, M.D., Vice-Chairman, will serve for the term January 1986 to December 1987.

(2) AMA Delegation Participation

in Regional Caucus . . . Noted that the New Jersey Delegation has accepted the opportunity to join the Great Lakes Coalition (composed of the states of Indiana, Illinois, Michigan, New York, Pennsylvania, and Ohio).

Report of Executive Director . . .
(1) MSNJ 1985 Paid Membership . . . Noted that by year-end, the total 1985 paid membership will equal the 1984 paid membership.
(2) Review of Financial Statements . . . Approved the financial statements for the period ending November 30, 1985.
(3) NJ State Medical Underwriters, Inc. . . . Noted that the 27 percent across-the-board rate increase will become effective with February 1, 1986, renewals.
(4) Repeal of Certificate of Need Law . . . Noted that a preliminary report shows that the management and trustees of New Jersey hospitals favor retaining the certificate of need law.
(5) Regulation . . .
(a) Physical Therapy Proposal . . . Noted again MSNJ's vigorous opposition to the proposal of the Physical Therapy Board.
(b) Nursing Supervision and Management . . . Noted again MSNJ's vigorous opposition to the Board of Nursing proposal.
(6) Request for MSNJ To Appear Amicus . . . Noted that MSNJ will not join amicus in a professional liability case because, as Mr. Maressa stated, punitive damages are inappropriate in a situation involving a nonintentional tort.

UMDNJ . . . Received the report by UMDNJ President Stanley S. Bergen, Jr., M.D., and noted the following new items: 1) the entire issue of UMDNJ's first bonds were sold; 2) Sidney Pestka, M.D., has been appointed chairman of the department of microbiology; and 3) the need for increased encouragement for blood donations.

NJ Hospital Association . . .
(1) Uncompensated Care . . . Noted that a proposed bill calls for assessment on hospital revenues and an increase in the so-called nuisance taxes.
(2) Physician Billing of Indigent Patients . . . Noted a suggestion from the Assistant Commissioner of Health stating that hospitals develop a system which would inform the physician that a patient has been declared indigent; inference is that once a patient is declared indigent, the physician will not bill for services rendered.
(3) Board of Nursing Litigation . . . Noted that NJHA is seeking judicial review of the Board of Nursing's attempts to regulate all unlicensed health care professionals through its proposed delegation of nursing tasks regulation.

Council on Medical Services . . .
(1) Seminars . . . Authorized a series of seminars dealing with HMOs, IPAs, PPOs, and other health care delivery systems.
(2) Prevention of Windfall Profits . . . Adopted the following amended recommendation:

> That MSNJ request legislation or regulation which would require insurance companies to reimburse patients equally, whether their physicians participate in a particular program or not.

(3) Development of Relative Value Guidelines . . . Approved the following recommendation:

> That the Board of Trustees postpone development of Relative Value Guidelines until a report is released by the AMA on the Health Care Financing Administration project.

(4) Capitation . . . Approved the following recommendation:

> That the subject of capitation be referred to the Ad Hoc Committee on Health Care Reimbursement Policies for study and recommendation.

Committee on Annual Meeting. . . Asked that the following items be placed on the agenda for the next Committee meeting: 1) discussion of a midweek annual meeting and the results of a county medical society poll; and 2) the possibility of holding more than one business meeting a year. Also, approved the cost breakdown for the Annual Meeting package. And, approved the following recommendation:

> That the Board of Trustees approve "Corporate Invasion in the Practice of Medicine" as the topic for the program scheduled for 8:30 A.M., Sunday, May 18, 1986, at The Westin Bellevue Stratford, in Philadelphia, Pennsylvania.

The Peer Review Oversight Committee . . .
(1) Committee Name . . . Approved the following recommendation:

> That the name of the PRO Oversight Committee be changed to the Peer Review Oversight Committee.

(2) PRO Criteria Committee . . . Noted that PRO of New Jersey, Inc., is in favor of appointing a representative of the MSNJ Committee to their PRO Criteria Committee.
(3) MSNJ Observer To Attend PRO Board Meetings . . . Approved the following recommendation:

> That the Board of Trustees again request PRO of New Jersey, Inc., to allow an MSNJ observer at PRO Board meetings.

(4) 1985 Annual Meeting Resolution . . . Approved the following recommendation:

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That a letter be written by the Board of Trustees to the PRO expressing dissatisfaction with their criteria as well as their method of implementation, and asking that a meeting between PRO of New Jersey, Inc., and MSNJ be set up for the purpose of collaborating on a criteria program which would be acceptable to New Jersey physicians.

Old Business . . .
Second Level Appeals—NJ DRG Program . . . Concurred with the action of the Executive Committee that advised the Commissioner of Health that MSNJ will not take part in the new structure of the second level patient appeal mechanism.

New Business . . .
(1) Petition for House of Delegate Seating . . . Postponed final decision on the request of the Society of Surgeons of New Jersey for seating in the MSNJ House of Delegates; the Society of Surgeons has 143 members and MSNJ bylaws state membership must be 200.
(2) Resolution To Designate Blood a Service . . . Postponed action on the resolution from the Essex County Medical Society requesting MSNJ petition the governor and the legislation to adopt legislation designating blood a service and not a product.
(3) AIDS . . . Referred to the Council on Public Health the need to address the issue of AIDS.

UMDNJ Notes

Stanley S. Bergen Jr., M.D.
President

From time to time, I like to spotlight some of the basic and clinical science research underway at the University's schools. This month, I'll focus on two projects at UMDNJ-Rutgers Medical School.

Michael Lewis, Ph.D., professor and director of the gifted clinic at the medical school's Institute for Study and Child Development, reports that certain sleeping patterns may be among the signs that a child is gifted; and in an unrelated project, Richard A. Parisi, M.D., an assistant professor of medicine, has found a link between the large muscle of the tongue and the onset of sleep apnea.

Dr. Lewis—with the aid of colleagues Carolyn McGuffog, Ph.D., and Candice Feiring, Ph.D.—found that many gifted children between one-and-a-half and four years of age have trouble falling asleep easily, wake early, or are unable to sleep for long periods. Explaining that the findings are preliminary, the researcher said he is unsure why proficiency may manifest different sleeping patterns. He theorizes that perhaps brain functions of gifted children are different, or that they are unusually attentive to their environment and want to stay awake and "take it all in." Dr. Lewis, who has a national reputation on the subject of gifted children, said that in the legal definition, "giftedness" means a child has taken an IQ test and has scored between 130 and 135, although some researchers set the mark higher. However, a single reading does not necessarily reflect a child's ability. Another sign of giftedness is a child having unusual skills and capacities, whether in one area or several. After evaluating 150 children at the Institute, Drs. Lewis, McGuffog, and Feiring have developed a list of signs of potential giftedness in infants and toddlers, including: alertness and attentiveness; an early fear of strangers; and early language use. One final point on giftedness and various sleeping patterns: Dr. Lewis notes that while these children give the impression they are having sleeping problems, this is not the case. The real problem is that parents do not get enough sleep as a result.

In the other research project, involving a very real and dangerous sleeping problem, Dr. Parisi has found that the failure of the large muscle of the tongue to contract during a certain deep sleep phase causes it to collapse against the back of the airway, blocking the passage. The result is the condition we call sleep apnea. The studies have earned the young researcher a $304,000 grant from the National Institutes of Health to further the investigation. Dr. Parisi has found that the tongue's large muscle fails to contract during the Rapid Eye Movement (REM) phase of sleep—also known as the dream phase—allowing the airway to close and breathing to stop.

In the studies with adult male goats, Dr. Parisi has found that the tongue, instead of contracting with the diaphragm, falls back into—and obstructs—the throat. Goats are used in the study, Dr. Parisi explains, because they are docile, easy to handle, and their pulmonary system is similar to that of man. The animals, whose days and nights are reversed artificially in their living environment, are quite content to sleep during the day while their oxygen levels and breathing are monitored via implanted electrodes. It was in this way that it was learned the tongue was not contracting during sleep.

Although labeling the preliminary results "an encouraging find," Dr. Parisi (who is working under the sponsorship of Norman Edelman, M.D., chief of the division of pulmonary and critical care medicine at the medical school, and Teodoro V. Santiago, M.D., associate professor of medicine) explains that he and his colleagues have much more to learn about the basic physiology of breathing during sleep.

UMDNJ is only one of a few medical centers around the country investigating this sometimes life-threatening condition that primarily affects adult males over 30 years of age who snore. Obesity and advanced age also are regarded as risk factors. Alcohol and drugs—whether by prescription or not—can worsen the condition.

MSNJ Auxiliary

Bernardine Maloney
President

To be charged with malpractice has been likened to the pain of a death in the family. Reactions include utter disbelief, shock, and confusion. As reassuring as family members may be, they also are victims of shock.

Following the shock, might come feelings of guilt, as in a death, that more could have been done. Anger quickly follows—anger at the person who has caused the situation, anger at the situation itself, and anger at the world in general.

Finally, comes the realization that the situation must be dealt with; it is time to face the reality and deal with it.

Throughout this process the problems and stresses are many and have a profound effect on the physician, the physician's spouse, and even the children. In investigating various approaches to "malpractice stress syndrome," it appears that the "state of the art" is to create local peer support groups.

This is just one issue with which the Auxiliary deals. Contact us for more information at 609-896-1766.

AMNJ Report

Dennis R. Filippone, M.D.
President

As we enter 1986, the Academy looks forward to celebrating its 75th anniversary of service to the physicians of New Jersey. A number of activities are in the planning stages including a jointly sponsored lecture with the Medical History Society of New Jersey and the UMDNJ-George F. Smith Library. This year also marks the 15th anniversary of AMNJ's library contribution to UMDNJ which included our valuable rare book collection.

The Academy's newly created Long-Range Planning Committee held a workshop on December 11, 1985, at the Loew's Glenpointe Hotel in Teaneck. The Committee and an outside consultant experienced in medical association promotion discussed the concept of marketing and how the Academy can offer our members more benefits in this area.

The Academy has been asked to participate in The Governor's Jersey Pride Awards Project for 1986. The goal of this effort is to establish an annual series of awards to present to New Jerseyans for outstanding achievement in their fields. This project is part of an overall public relations effort to improve the image of New Jersey. A total of 16 awards will be presented of which the Governor's Selman Waksman Medicine Award is included. The development of criteria for the award and a nomination and selection process as well as a date are being discussed.

The Academy presently is becoming involved with computers and two objectives have been established. First, the current word processing system at the Executive Offices is to be upgraded to become a database management system. Second, a member service involving computers is to be investigated. The internal data processing needs of Academy operations, the first objective, is underway and a four-computer network has been installed at the Executive Offices, which provides a comprehensive database management system. The member service aspect of the Academy's computer objective is getting underway. Vendors of hardware and software are being compiled, computer consultants are being scrutinized, and a preliminary survey of AMNJ Fellows is complete. A subcommittee of the Education Committee will review and discuss options for member services in the near future.

Those member services presently being considered are a computer consultation service and a computerized bulletin board. Several requests have come to the Academy from physician practices around the state. These requests are from groups planning to install or upgrade computerized practice management functions. Several consultant referrals successfully have been made. It is likely this service will grow as more practitioners consider the benefits of practice automation. A computer bulletin board also is under consideration and could be operational as early as spring 1986. This service could: provide information on AMNJ programs, include computer newsletters, open access for physicians to leave and receive messages, and offer electronic drug reference. The physician could access this bulletin board.

The coming year should be exciting for members interested in the

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Academy's growing computer services.

New Members

NEW JERSEY MEDICINE would like to welcome the following new members to the Medical Society of New Jersey:

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Jacob L. Levin, M.D., Linwood
Claudia Prapawiwat, M.D., Pomona

Bergen County
Wayne J. Altman, M.D., Rutherford
James E. Buirkle, M.D., Oradell
Mardiros Derbarghamian, M.D., Ridgefield
Henry J. Rose, M.D., Rutherford

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Robert S. Altin, M.D., Browns Mills
David F. Greenbaum, M.D., Willingboro
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Roy Puskas, M.D., Marlton

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Mercer County
Enrique Alvarez, M.D., Lawrenceville
Harold J. Brown, M.D., Trenton
Lucius D. Clay, III, M.D., Princeton
Jeffrey L. Lipkowitz, M.D., Lawrenceville
Michael A. Willen, M.D., Trenton

Middlesex County
Maurice Bienenfeld, M.D., North Brunswick
Robert Collier, M.D., Piscataway
Stephen S. Cook, M.D., New Brunswick
Benjamin Fand, M.D., Perth Amboy
Louis J. Flancbaum, M.D., New Brunswick
Charles Highstein, M.D., Edison
Zofia Hrymoc, M.D., North Brunswick
Susan A. McManus, M.D., Somerset
Victor M. Molina, M.D., New Brunswick
Robert M. Niemiera, M.D., Perth Amboy
Kasthuri Pothiraj, M.D., East Brunswick
Jose Sobrepena, M.D., Long Branch
Thomas E. Strax, M.D., Edison
Bernardo Toro-Echague, M.D., Plainfield

Monmouth County
Gaetano J. Aquilino, M.D., West Long Branch
Frank H. Arlinghaus, Jr., M.D., Rumson
Norman A. Back, M.D., Freehold
Salvatore A. Croce, M.D., Englishtown
Jeffrey S. Daniels, M.D., Oakhurst
Tarsem Gupta, M.D., Neptune
Ahmad J. Haddad, M.D., Red Bank
Ann M. Hughes, M.D., Oakhurst
Gabor Kovacs, M.D., Englishtown
Steven L. Oreck, M.D., Oakhurst
Bernadette M. Weiss, M.D., Long Branch

Morris County
Patricia Dreyfuss, M.D., Boonton
Jerrold B. Goldstein, D.O., Florham Park
James J. Kuchera, M.D., Morristown
Peter N. Thompson, M.D., Chatham
Douglas M. Weissman, M.D., Morristown
Alfred F. Weitzman, M.D., Morristown

Passaic County
Les A. Burns, M.D., Wayne
David A. DeBell, M.D., Passaic
Claire B. Gonsalves, M.D., Clifton
Leonard Joachim, M.D., Paterson
Daniel H. Levin, M.D., Clifton
Ronald R. Majka, M.D., Clifton
Leonard T. Nicosia, M.D., Wayne
Jaiminikumar R. Patel, M.D., Lodi
Alan M. Tell, M.D., Fair Lawn

Somerset County
Christine A. Ganitsch, M.D., Basking Ridge
James J. Hutchins, M.D., Somerville
Gaston G. Trigos, M.D., Somerset

Union County
Joel Confino, M.D., Westfield
Lawrence J. DeAngelis, M.D., Springfield
David E. Guyer, M.D., Union
Neil B. Horner, M.D., Mountainside
Anandhi M. Murthy, M.D., Elizabeth
Paul Schackman, M.D., Madison
Daniel J. Thomas, M.D., Madison
Carl J. Vitale, M.D., Union

Warren County
Marc N. Abo, M.D., Phillipsburg

Physicians Seeking Location in New Jersey

The following physicians have written to the Executive Offices of MSNJ seeking information on possible opportunities for practice in New Jersey. The information listed below has been supplied by the physician. If you are interested in any further information concerning these physicians, we suggest you make inquiries directly to them.

ANESTHESIOLOGY—S.H. Emami Kelishadi, M.D., 4201 Cathedral Ave., NW 222E, Washington, DC 20016. Tehran (Iran) 1957. Also, emergency medicine. Group or partnership. Available.

EMERGENCY MEDICINE—S.H. Emami Kelishadi, M.D., 4201 Cathedral Ave., NW 222E, Washington, DC 20016. Tehran (Iran) 1957. Also, anesthesiology. Group or partnership. Available.

Julian A. Sellek, M.D., 1501 South Greenway, Coral Gables, FL 33134. Salamanca (Spain) 1963. Also, family practice. Board eligible. Board certified (FP). Group, solo, partnership. Available.

FAMILY PRACTICE—Lawrence S. Gordon, M.D., 1016 Spinnaker Way, Apt. G-1, Melbourne, FL 32935. Rutgers 1982. Board certified. Group, partnership, solo. Available spring 1986.

Debra T. Miller, M.D., 1428 D Mews Dr., Kansas City, MO 64131. Hahnemann Medical School 1982. Board eligible. Group or partnership. Available.

Jeffrey C. Schultz, M.D., 301 Victoria Dr., Monroeville, PA 15146. University of Massachusetts 1983. Board eligible. Group. Available July 1986.

Julian A. Sellek, M.D., 1501 South Greenway, Coral Gables, FL 33134. Salamanca (Spain) 1963. Also, emergency medicine. Board certified. Board eligible (EMER). Group, solo, partnership. Available.

Susan F. Stickels, M.D., 1913 E. Kenmore Pl., Shorewood, WI 53211. Board certified. Group or HMO. Available.

Lew Herbert Valliant, M.D., 115 Mountain View Dr., Elkins, WV 26241. Queen's University (Canada) 1976. Board certified. Solo or group. Available.

INTERNAL MEDICINE—Walter M. Bakun, M.D., 199 Casterline Rd., Denville, NJ 07834. St. George's University (Grenada) 1983. Board eligible. Group, partnership, solo. Available July 1986.

Joseph Cohen, M.D., 16 Mountainbrow Village, Corning, NY 14830. New York Medical College 1982. Board eligible. Group. Available July 1986.

Arkady S. Fishman, M.D., 55 Nagle Ave., Apt. 1E, New York, NY 10040. Aktyubinsk Medical Institute 1969. Board certified. House · staff, group, nursing home. Available.

Serge I. Kaftal, M.D., 125 Gates Ave., Apt. 38, Montclair, NJ 07042. Lodz Medical Academy (Poland) 1978. Solo, group, health center service. Available.

H. Homayouni, M.D., 459 Hamilton Pl., Hackensack, NJ 07601. Tehran (Iran) 1971. Board certified. Partnership. Available.

Marian Lynn Taylor, M.D., 7 Loriann Rd., Warren, NJ 07060. UMDNJ 1983. Board eligible. Group or partnership. Available June 1986.

Dipak Vora, M.D., 509 Indian Creek Dr., Wilkes-Barre, PA 18702. University of Bombay (India). Board eligible. Available July 1986.

OPHTHALMOLOGY—Kenneth R. Brein, M.D., 55 Beacon St., Pittsburgh, PA 15217. University of Pennsylvania 1982. Board eligible. Group, partnership, solo. Available July 1986.

Andrew R. Gewirtz, M.D., 64 Pond Rd., Apt. G3, Woodbury, NY 11797. Chicago 1978. Board eligible. Board certified (PED) Single or multispecialty group. Available.

RADIOLOGY—Jesus S. Santos, Jr., M.D., RD 2, Gibson Rd., Goshen, NY 10924. Santo Tomas (Philippines) 1960. Board certified. Available.

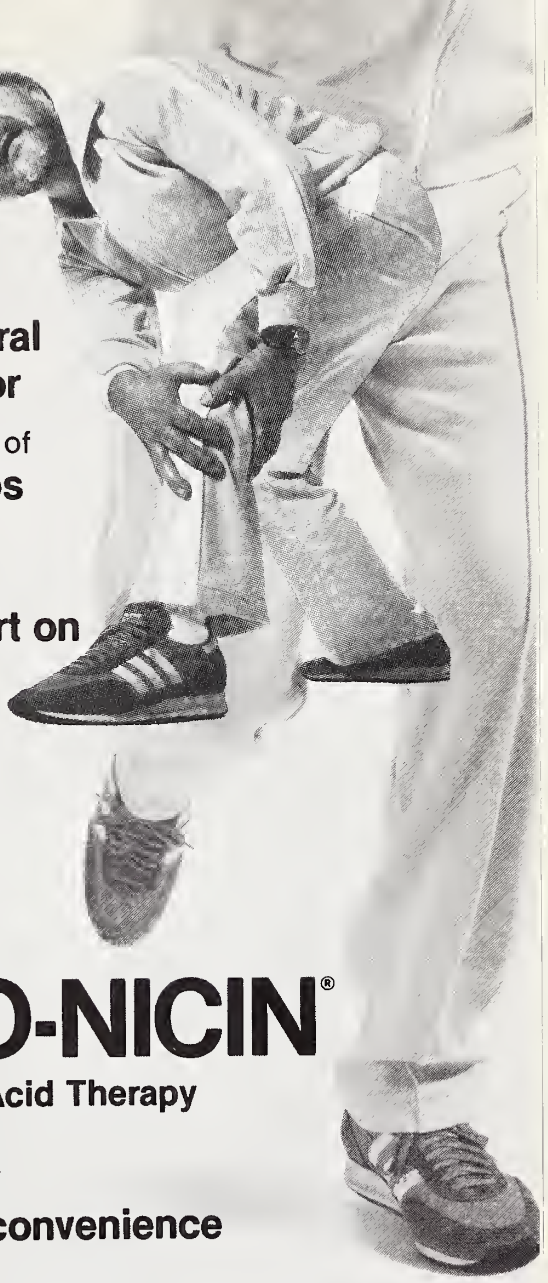
SURGERY—Derrick John Martin, M.D., 13-2 Granada Cres., White Plains, NY 10603. West Indies 1977. Board certified. Group, partnership, solo. Available.

Niranjan V. Rao, M.D., 3311 Clarendon Rd., Cleveland Heights, OH 44118. M.R. Medical College (India) 1976. Board certified. Also, vascular surgery. Group, partnership, solo. Available.

UROLOGY—Louis C. Galdieri, M.D., 1563 Hampton Knoll Dr., Akron, OH 44313. Guadalajara (Mexico) 1979. Group or partnership. Available August 1986.

John R. Scheuch, M.D., 116 Sycamore Rd., Jersey City, NJ 07305. St. George's University (Grenada) 1981. Board eligible. Partnership or group. Available July 1986.

Edward C. White, M.D., 5 Starlight Ct., North Babylon, NY 11704. Guadalajara (Mexico) 1979. Board eligible. Solo, partnership, group. Available.



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Indications: For use as a vasodilator in the symptoms of cold feet, leg cramps, dizziness, memory loss or tinnitus when associated with impaired peripheral circulation. Also provides concomitant administration of the listed vitamins. The warm tingling flush which may follow each dose of LIPO-NICIN® 100 mg. or 250 mg. is one of the therapeutic effects that often produce psychological benefits to the patient.

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CARDIOLOGY UPDATE. . .

IS DESIGNED FOR THE PHYSICIAN AND PROVIDES AN INTENSIVE SURVEY OF THE CURRENT STATUS OF CLINICAL CARDIOLOGY. . .

WEDNESDAY, MARCH 5, 1986

MITRAL VALVE PROLAPSE

MODERATOR: JOEL MORGANROTH, M.D.

3:00	CASE PRESENTATION: CHEST PAIN	*Edward Catherwood, M.D.*
3:30	DISCUSSION, INCLUDING WORK-UP AND MANAGEMENT	*William S. Haaz, M.D.*
4:00	CASE PRESENTATION: ARRHYTHMIAS	*Joseph R. Carver, M.D.*
4:30	DISCUSSION, INCLUDING WORK-UP AND MANAGEMENT	*Allan M. Greenspan, M.D.*
5:00	QUESTIONS AND ANSWERS	*The Audience*

LECTURE HALL "A"—2nd floor New College Building, Hahnemann University
15th and Vine Streets, Philadelphia, PA

• NO REGISTRATION FEE • NO ADVANCE REGISTRATION REQUIRED •
• CME CATEGORY I CREDITS CERTIFIED •
WINE & CHEESE SERVED FOLLOWING CONFERENCE

CME CALENDAR

The following is a list of continuing medical education courses for the next two months. Contact the sponsoring organization for further information.

This list is compiled through the cooperation of the Committee on Medical Education of the Medical Society of New Jersey, The Academy of Medicine of New Jersey, the New Jersey Chapter of the American Academy of Family Physicians, and the Office of Continuing Medical Education of the UMDNJ. For information on accreditation, please contact the sponsoring organization(s), indicated by italics—last line of each item.

OBSTETRICS/GYNECOLOGY

March

18 **Drugs and the Placenta**
8:30-9:30 A.M.—Newark Beth Israel Medical Center
(Newark Beth Israel Medical Center and AMNJ)

21 **Semmelweis-Waters Ob/Gyn**
22 **Conference**
23 Resorts International Hotel, Atlantic City
(UMDNJ-NJ Medical School)

PEDIATRICS

March

15 **Neonatal Surgical Emergencies**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

April

19 **Urologic Emergencies in the Newborn**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

RADIOLOGY

March

8 **Radiology of the GI Tract**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

SURGERY

March

4 **Surgical Grand Rounds**
11 7-9 A.M.—Hackensack Medical
25 Center, Hackensack
(Hackensack Medical Center and AMNJ)

April

1 **Surgical Grand Rounds**
8 7-9 A.M.—Hackensack Medical
15 Center
29 *(Hackensack Medical Center and AMNJ)*

22 **Neoplastic Diseases of the Lung**
8-10 P.M.—Englewood Club, 115 E. Palisade Ave., Englewood
(Englewood Surgical Society and AMNJ)

26 **Role and Operation of a Day Surgical Unit in a Tertiary Care Center**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

SURGICAL SPECIALTIES

March

15 **Neonatal Surgical Emergencies**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

April

Orthopedic Symposium
4 3:15-5:30 P.M.—Case Presentations, Marriott, Somerville
5 7:30 A.M.-4 P.M.—Rutgers Medical School, Piscataway
(UMDNJ and AMNJ)

April

Mini-Residency in Proctology and Diseases of the Colon
One Week Commitment—contact Overlook Hospital, Summit
(Overlook Hospital and AMNJ)

2 **Mini-Residency in Occupational**
18 **Medicine**
8:30 A.M.-4 P.M.—Contact UMDNJ-Rutgers Medical School, Piscataway
(UMDNJ and AMNJ)

2 **Common Skin Problems**
1:30-2:30 P.M.—Rutgers Community Health Plan, 57 U.S. Hwy #1, New Brunswick
(Rutgers Community Health Plan and AMNJ)

2 **Heart Failure**
9 **Diseases of the Pancreas**
16 **Inflammatory Bowel Disease**
23 **Principles of Drug Therapy and**
30 **Drug Interaction**
Bradyarrhythmia
8:30-10 A.M.—Alexian Brothers Hospital, Elizabeth
(Alexian Brothers Hospital and AMNJ)

2 **Medical Lecture Series**
9 10:30 A.M.-12 noon—Christ
16 Hospital, Jersey City
23 *(Christ Hospital and AMNJ)*
30

3 **Human Genetics**
10 4-6 P.M.—Institute for Medical
17 Research, Copewood St., Camden
24 *(Institute for Medical Research and AMNJ)*

4 **Major Advances in Cancer—1986**
8 A.M.-4 P.M.—NJ Medical School, Piscataway
(UMDNJ and AMNJ)

5 **Nutrition Support of Trauma Patients**
8 A.M.-4 P.M.—Marriott Hotel, Newark Airport
(UMDNJ and AMNJ)

8 **Advances in Allergy and Immunology 1986**
8:30 A.M.-1:30 P.M.—Holy Name Hospital, Teaneck
(Holy Name Hospital and AMNJ)

16 **AIDS and the IV Drug Abuser**
9 A.M.-4 P.M.—MSNJ Executive Offices, Lawrenceville
(NJ State Dept. of Health, MSNJ, and AMNJ)

16 **Dermatological Conference**
6-9 P.M.—Rutgers Community Health Plan 57 U.S. Hwy #1, New Brunswick
(UMDNJ and AMNJ)

17 **Current Concepts in the Prevention and Management of Osteoporosis**
5-6:30 P.M.—Somerset Medical Center, Somerville
(Somerset Medical Center and AMNJ)

19 **Urologic Emergencies in the Newborn**
26 **Role and Operation of Day Surgeon Unit in a Tertiary Care Center**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

NEUROLOGY/PSYCHIATRY

March

3 **Medication of a Borderline Psychiatric Patient**
8:15-10 P.M.—9 Marquette Road, Upper Montclair
(Essex Psychiatric Seminar and AMNJ)

6 **Case Seminars**
20 8-10 P.M.—312 Harding Drive, South Orange
(Advanced Psychiatric Study Group and AMNJ)

8 **Overview of Diagnosis and Treatment of Pathological Gambling**
9 A.M.-3 P.M.—Somerset Hilton, Somerset
(NJ Psychiatric Association and AMNJ)

The Academy of Medicine
of New Jersey
and
UMDNJ-New Jersey Medical School
present
A SYMPOSIUM ON

Management of Head Trauma in the 80's

on

SATURDAY, MARCH 15, 1986
8:00 A.M.-4:00 P.M.
at
NEW JERSEY MEDICAL SCHOOL, NEWARK

The purpose of this symposium is to provide an opportunity for health care providers to meet and discuss development and controversies in the rapidly changing field of Head Trauma Management.

For further information contact:

DEBORAH GRAY
EXECUTIVE OFFICES
The Academy of Medicine of New Jersey
Two Princess Road
Lawrenceville, New Jersey 08648
(609) 896-1717

The Academy of Medicine
of New Jersey
and
Radiological Society of New Jersey
present a
Major Symposium
ON
NEW FRONTIERS IN LIVER and BILIARY TRACT RADIOLOGY
on
Saturday, March 15, 1986
8:30 a.m.-4:00 p.m.
at
Newark Airport Marriott

Participants will be able to:

- Discuss advances in imaging technology which improve diagnostic capabilities of liver, gallbladder and biliary disease.
- Compare the role of conventional imaging with new imaging techniques in hepatobiliary disease.

For further information contact:

DEBORAH GRAY
EXECUTIVE OFFICES
The Academy of Medicine of New Jersey
Two Princess Road
Lawrenceville, New Jersey 08648
(609) 896-1717

THE LEGACY OF KAREN ANN QUINLAN
A TENTH YEAR RETROSPECTIVE.
Wednesday, March 26, 1986
8:30 A.M.-4:00 P.M.
RUTGERS MEDICAL SCHOOL
PISCATAWAY, NJ (Main Auditorium)

PRESENTED BY:
AMERICAN COLLEGE OF PHYSICIANS, NEW JERSEY CHAPTER
AMERICAN SOCIETY OF LAW AND MEDICINE
CITIZEN'S COMMITTEE OF BIOMEDICAL ETHICS
SOCIETY FOR HEALTH AND HUMAN VALUES
MEDICAL SOCIETY OF NEW JERSEY
UNIVERSITY OF MEDICINE AND DENTISTRY OF NEW JERSEY

Honorary Chairman: Thomas H. Kean, Governor of New Jersey

Panelists: George J. Annas, Christine K. Cassel MD, Eric J. Cassell MD, Hon. Richard J. Hughes, Joanne Lynn MD, Rev. John J. Paris, Mark Siegler MD, Alan J. Weisbard

Moderators: Paul Armstrong, Robert Cassidy, Russell L. McIntyre, Michael A. Nevins MD

Luncheon Speaker: Stanley S. Bergen MD

5 CME Category 1 Credits Approved

Registration $40.00 includes luncheon

For additional information contact Program Chairman:
Michael Nevins MD
595 Chestnut Ridge Road
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(201) 391-1078

Detach and mail to ACP, 4200 Pine Street, Philadelphia PA

Enclosed is my check for _____ payable to ACP

Name (please print) ____________________

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A confirmation will be sent to you along with a program and directions to Rutgers Medical School.

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of
Thomas Jefferson University
Departments of Psychiatry and Neurology and
The Office of Continuing Medical Education
presents

ALZHEIMER'S DISEASE:
A COMPREHENSIVE APPROACH

April 4, 1986
at the
Bellevue Straford Hotel
Philadelphia, PA

By means of lectures, group problem solving and panel discusssions physicians will have a better clinical understanding of the Recent Advances in Alzheimer's Disease and Related Disorders.

CME Credit: 6 hours in Category 1
6 prescribed hours in AAFP (pending)
6 prescribed hours in APA (pending)

For further information contact:
The Office of Continuing Medical Education
Jefferson Medical College
1025 Walnut Street, Room G3
Philadelphia, PA 19107
215 (928-6992)

April

1 **Abdicating Fathers, Homosexual Sons**
8:30-10:30 P.M.—Marriott Hotel, Saddle Brook
(NJ Council of Child and Adolescent Psychiatry and AMNJ)

3
17 **Case Seminars**
8-10 P.M.—312 Harding Drive, South Orange
(Advanced Psychiatric Study Group and AMNJ)

7 **Violence in a Marriage**
8:15-10:15 P.M.—39 Crescent Ave., Passaic
(Essex Psychiatric Seminar and AMNJ)

MEDICINE

March

Mini-Residency in Proctology and Diseases of the Colon
One Week Commitment—contact Overlook Hospital, Summit
(Overlook Hospital and AMNJ)

4 **Scleroderma and Variants**
7-8 P.M.—West Hudson Hospital, Kearny
(West Hudson Hospital and AMNJ)

5 **Hypercalcemia**
19 **Evaluation of Kidney Stones**
1:30-2:30 P.M.—Rutgers Community Health Plan, 57 U.S. Hwy. #1, New Brunswick
(Rutgers Community Health Plan and AMNJ)

5 **Acute M.I.—Reperfusion**
12 **Vasculitis—SLE**
19 **Approach to Treatment of Rheumatoid Arthritis**
26 **Reiter's Syndrome, Psoriatic Arthritis and Arthritis Associated with Gastrointestinal Disease**
8:30-10 A.M.—Alexian Brothers Hospital, Elizabeth
(Alexian Brothers Hospital and AMNJ)

5 **Adolescent Medicine**
6-8 P.M.—NJ Medical School, Newark
(UMDNJ)

5 **Adolescent Medicine**
6-8 P.M.—NJ Medical School, Piscataway
(UMDNJ)

5 **Adolescent Medicine**
6-8 P.M.—Halloran Plaza, Rt. 130 North, Pennsauken
(UMDNJ)

5 **Medical Lecture Series**
12 10:30 A.M.-12 noon—Christ
19 Hospital, Jersey City
26 *(Christ Hospital and AMNJ)*

6 **Human Genetics**
13 4-6 P.M.—Institute for Medical
20 Research, Copewood St., Camden
(Institute for Medical Research and AMNJ)

11 **Postmenopausal Osteoporosis**
18 **Atypical Pneumonia: Hidden Hazards**
9-10 A.M.—Holy Name Hospital, Teaneck
(Holy Name Hospital and AMNJ)

19 **Approach to the Febrile Neutropenic Patient**
1-2 P.M.—West Hudson Hospital, Kearny
(West Hudson Hospital and AMNJ)

19 **Dermatological Conference**
6-9 P.M.—Rutgers Community Health Plan, 57 U.S. Hwy. #1, New Brunswick
(UMDNJ and AMNJ)

20 **Prostaglandins in Gastrointestinal Disease**
11 A.M.—St. Joseph's Hospital and Medical Center, Paterson
(St. Joseph's Hospital and Medical Center and AMNJ)

20 **Occupational Reproductive Hazards**
5-6:30 P.M.—Somerset Medical Center, Somerville
(Somerset Medical Center and AMNJ)

26 **I.A.B.P. and Medicine in China**
8-10 P.M.—Englewood Club, 115 East Palisade Avenue, Englewood
(Englewood Surgical Society and AMNJ)

COMMON CLINICAL CHALLENGES IN THE ELDERLY
A POSTGRADUATE COURSE

Presented by
Philadelphia Geriatric Center
and the Medical College of Pennsylvania

Sponsored by the
American College of Physicians

APRIL 3-5, 1986
The Wyndham Franklin Plaza
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Philadelphia, PA 19103

Common Clinical Challenges in the Elderly is designed to provide up-to-date information for the clinician who cares for the elderly in either ambulatory or inpatient settings. It is recommended specifically for internists, family practitioners and general practitioners.

Topics will include: GI disorders, cardiovascular diseases, infections, musculoskeletal disease, endocrine disorders, altered mental status, sleep disorders, foot problems, practical applications of exercise physiology, and drug prescribing.

Participants will receive 18-1/3 hours of Category I credit in Continuing Medical Education. (Reviewed and acceptable for 18 prescribed hours by the AAFP.)

For more information contact:
Ms. Nancy Spaw
Department of Medicine
Philadelphia Geriatric Center
5301 Old York Road
Philadelphia, PA 19141
(215) 456-2943

Philadelphia Geriatric Center

ANNUAL CME ASSEMBLY
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MEDICAL SOCIETY OF THE
STATE OF NEW YORK

MAINTAINING THE QUALITY OF
HEALTH CARE INTO THE 21st CENTURY
MARCH 7-9, 1986
THE NEW YORK HILTON, NEW YORK CITY
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HIGHLIGHTS

22 CME Sessions; Panel Discussions; Technical Exhibits: two Receptions sponsored by MSSNY: President's Dinner Dance.

Registration: There is no fee for members of the New Jersey State Medical Society, residents, medical students, or medical assistants. Non-member physicians—$50; Allied Professions $10.

☐

For a copy of the preliminary program, contact: MSSNY, 420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.
Attn: Division of Meeting Services
516-488-6100

Resident Association
of
The Medical Society of New Jersey
presents

PREP FOR PRACTICE

A PRACTICE MANAGEMENT SEMINAR FOR RESIDENTS
SATURDAY, MARCH 1, 1986
9:00 A.M.-4:30 P.M.
at the Executive Offices of the
MEDICAL SOCIETY OF NEW JERSEY
Lawrenceville, New Jersey 08648

MORNING PROGRAM

9:00-9:15	Introduction
9:15-10:00	**Search for the Right Opportunity** • How the Changing Complexion of Health Care Will Affect You • Professional Consideration in Selecting a Site • Personal Considerations • Financial Considerations
10:00-10:15	Questions and Answers
10:15-11:15	**Types of Practice** • Solo • Partnership • Group • HMOs
11:15-11:30	Questions and Answers
11:30-12:00	**Physical Facilities** • Space Requirements • Floor Plans • Patient and Work Flow
12:00-12:15	Questions and Answers
12:15-1:00	Lunch

AFTERNOON PROGRAM

1:00-1:30	**Business Insurance** • Types • How Much and When
1:30-1:45	Questions and Answers
1:45-2:30	**Personnel** • What You Need • How to Find • Job Descriptions • Performance Reviews and Salary Reviews
2:30-2:45	Questions and Answers

2:45-3:15	**Systems**
	• Time Management
	• Charts and Records
	• Phones
	• Billing, Insurance and Collection
	  • Fees
	  • Medicare

3:15-3:30	Questions and Answers
3:30-4:15	**Financial Management**
4:15-4:30	Questions and Answers
4:30	Adjournment

FACULTY

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LETTERS TO THE EDITOR

Jewish Views of Life and Medical Practice

October 23, 1985

Dear Doctor Krosnick:

The article, "Selected Jewish Views of Life and Medical Practice" by Lindenthal, Michelson, and Schlesinger, (*J Med Soc NJ* 82: 795-799, 1985) does not represent as clearly as it might the history and basic viewpoint of conservative Judaism.

In the late 19th century, Jews found themselves under increasing pressure to adapt their way of life to their growing participation in society outside the Ghetto. Among responses to this pressure was the growth reform movement which had its beginnings in Germany and then spread to America. In 1886, a meeting of reformed Jews was held in Pittsburgh. It concluded by rejecting many of the traditional Jewish practices and, in essence, the historically continuous process of Rabbinic interpretation of the Torah by which these practices had grown and been modified through the ages. In the following years, many of those present at the Pittsburgh meeting who were unwilling to participate in this radical departure from tradition founded the Jewish Theological Seminary of America, the conservative Rabbinic seminary. In 1913, Solomon Schechter, the first leader of the Seminary, founded the United Synagogue of America, a lay organization for the promotion of conservative Judaism. The objective of this organization is "to assert and establish loyalty to the Torah and its historical expositions . . ." [from the Preamble to the Constitution of the United Synagogue of America].

Thus, in conservative Judaism, as in orthodox, the process of growth and change should be emphasized to be seen in the context of thousands of years of Rabbinic tradition in the interpretation of the Torah, not as a matter of contemporary "legislation." Conservative, not reform, Judaism truly has its "roots" in American soil and was founded more in response to the reform than to the orthodox movement.

(signed) Frederick J. Stone, M.D.

November 26, 1985

Dear Doctor Krosnick:

In his letter to the Editor, Dr. Stone has pointed out the opposition of the very early founders of conservative Judaism to the reform movement. This view echoes the outlook of Professor Salo Baron, who first suggested it a half-century ago.[1]

Dr. Stone's opinion of the role of Halacha among conservative Jews, however, is misleading if it characterizes their view in any blanket fashion. In addition to the events of recent history and a considerable literature that refutes his position, there is enlightenment in a recent volume published by the United Synagogues of America in celebration of the 70th anniversary of its founding by Solomon Schechter. The chapter, "Halakhah-Conservatism and Jewish Law," written by the author, a scholar of conservative Judaism and who is ordained by the Jewish Theological Seminary, reviews the attitudes concerning the Halacha held by the Conservative Movement. Readers will draw obvious conclusions. Those of the author are as follows: "The above brief survey of the handling of the question of Halakah in the conservative movement should provide convincing testimony to the place that the subject has held in the evolution of the movement. It would seem that the large majority of conservative leaders are quite gratified at the fact that the Committee on Jewish Law and Standards has finally come to 'legislate,' rather than merely 'interpret,' as was the case for so many frustrating years. Whether this legislation will ultimately be implemented only within the conservative movement, or will be accepted by a broad new international consensus, remains a question that only time will decide."[2]

Although this clear statement regarding the acceptance of legislation over mere interpretation reveals Dr. Stone's mistaken charge, he has also missed the point and purpose of the article as a whole. The points he raises veer from the essential purpose of the article.

(signed) Jacob Jay Lindenthal, Ph.D.

REFERENCES

1. Baron SW: *A Social and Religious History of the Jews.* New York, NY, Morningside Heights, Columbia University Press, 1937, pp. 257-258.

2. Rosenblum H: *Conservative Judaism: A Contemporary History.* New York, NY, United Synagogue of America, 1983, p. 126.

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BOOK REVIEWS

Pediatric Nuclear Medicine; Viral Diseases of the Eye

Pediatric Nuclear Medicine

S.T. Treves. New York, NY, Springer-Verlag, 1985. Pp. 338. Illustrated. ($74.50).

Pediatric Nuclear Medicine is a comprehensive review of multiple areas of pediatric medicine where nuclear medicine diagnostic procedures make an important contribution. The book is written from the point of view of an experienced nuclear medicine clinician in an academic environment—Harvard Medical School.

Dr. Treves, editor of the book, is the co-author of all but one chapter. Therefore, the author delivers a coherent, cognitive approach to physiologic diagnosis in broad areas of pediatric medicine. There is an excellent discussion of radiation dose in each study with direct comparison to standard radiographic procedures. He clearly demonstrates, in each chapter, that the risk/benefit ratio for nuclear medicine procedures is clearly maximized compared to standard radiographic procedures.

There are 17 chapters ranging in subjects from bone, bone marrow, thyroid, and lung with excellent reproductions of scintigraphy and relevant clinical discussion.

This book is an excellent reference for the general pediatrician as well as the experienced radiologist and certainly for the nuclear medicine specialist. I believe the book demonstrates that pediatric nuclear medicine is a distinct subspecialty.

Louis S. Zeiger, M.D.

Viral Diseases of the Eye

Richard W. Darrell, M.D., (ed). Philadelphia, PA, Lea and Febiger, 1985. Pp. 341. Illustrated. ($46.50)

Ocular virology is an area that previously has not been covered indepth in any single volume. This current monograph presents aspects of this area not generally available elsewhere. An international selection of 33 authors has contributed indepth coverage on the pathogenesis of viral disease, mechanisms of latency in infection, resistance and immunity, clinical treatment, laboratory methodology, and epidemiology.

While this volume is of primary interest to the ophthalmologist in practice or training, it would be of value to practitioners of general medicine and pediatrics, virology, and oncology, if judged only by the variety of contributors. Contemporary problems of general interest include acquired immune deficiency as it relates to several other viruses such as cytomegalovirus and progressive multifocal leukoencephalopathy (Papovaviridae).

Fascinating chapters are devoted to the relationship of measles viremia and malnutrition with post-measles blindness and immunologic deficiency, and to a profound discussion of the general role of viruses in tumor production, using ocular tumors as the examples.

After an introductory taxonomy of viruses, most of the viral families are discussed. This introduction contains good tables summarizing the characteristics of pertinent virus families and their respective eye diseases, integrating and amplifying various viral manifestations.

The overall discussion of the classification, pathogenesis, and therapy of ocular Herpes simplex also is presented clearly. In addition to the characteristic lid and corneal lesions, a chapter is devoted to the less-recognized forms of Herpes simplex virus infection, follicular conjunctivitis and keratoconjunctivitis. This generally unappreciated topic is an interesting reawakening of the important differential diagnosis of presumed adenoviral disease.

A fine overview of the advances in the field of Varicella-Zoster research that have been made in the past 20 years, some of which will make it more possible to control severe Varicella-Zoster infections is presented. One of these four chapters provides extensive background laboratory research information, far exceeding immediate clinical application, thereby frequently obscuring relevant clinical data.

Adenoviral infections can be diagnostic and therapeutic dilemmas. The impression created here is that the clinical diagnosis may be derived with moderate specificity despite the problems in laboratory diagnosis.

Initially, one may question the value of the devotion of over 12 pages to the epidemiology of viruses causing acute hemorrhagic conjunctivitis; however, the background is necessary for this new entity, first recognized in 1969. The coxsackievirus A24 and enterovirus 70 mutated from domestic animal viral pools in Africa. This entity is unique in its geoepidemiology and neurologic sequelae.

Rubella, known for its acute manifestations, also is shown to have delayed effects, ocular and systemic, as a slow virus.

Since this book is an anthology, at times it suffers from repetition of the same material in different chapters, occasionally with an opposing viewpoint. Additionally, greater use could be made of line drawings and flow charts. Photographs in many cases are not illustrative, and are absent from chapters which would benefit from them. Abbreviations are employed excessively.

This book fills a great need, and should be read by every ophthalmologist and others involved with virology.

Michael L. Mund, M.D.

OBITUARIES

Drs. Connell; Czepiel; Gáspár; Gindhart; Gonzalez; Harz; Kennedy; Lee; Lopez; Starr

Dr. James V. Connell

A member of our Camden County component, James Vincent Connell, M.D., died on September 8, 1985, at the age of 72. A native of Philadelphia, Pennsylvania, Dr. Connell received his medical degree from Jefferson Medical College in that city, in 1942. He practiced general medicine in Gloucester City, and had been affiliated with Cooper Medical Center, Camden. Dr. Connell was a member of the American Medical Association. During World War II he served in the medical corps of the United States Navy.

Dr. Stanley J. Czepiel

The former chief of pathology at Englewood Hospital, Stanley John Czepiel, M.D., died at that hospital on August 27, 1985, at the age of 68. Born in Utica, New York, Dr. Czepiel received his medical degree at the University of Basel, Switzerland, in 1952. He was a member of our Hudson County component and of the American Medical Association.

Dr. István A. Gáspár

A former director of pathology and laboratory departments at Englewood Hospital, István Antal Gáspár, M.D., died on September 6, 1985, at the remarkable age of 87. A native of Hungary, Dr. Gáspár was graduated from the University of Budapest Medical School in 1923. He emigrated to the United States in 1927 and became director of the pathology department at Rochester (New York) General Hospital. In 1934 he founded one of the first schools for laboratory technicians, and joined the staff at Englewood Hospital in 1948. Dr. Gáspár was a member of our Bergen County component and of the American Medical Association. He was a Fellow of the College of American Pathologists, and was board certified in his specialty. Dr. Gáspár had written a number of scientific articles, some of which appeared in this journal. In 1973, he was a recipient of MSNJ's Golden Merit Award, honoring his 50 years as a physician.

Dr. Thomas D. Gindhart

A former member of our Mercer County component, Thomas David Gindhart, M.D., died on September 7, 1985, at his home in Middletown, Maryland, at the untimely age of 38. A native of Trenton, Dr. Gindhart received his medical degree at Hahnemann Medical College and Hospital, Philadelphia, in 1972. He had been a cancer expert at the National Institutes of Health, Bethesda, Maryland, made many contributions to the cause and prevention of the disease, and had presented papers both nationally and internationally.

Dr. Mario O. Gonzalez

Mario Olegario Gonzalez, M.D., a retired member of our Hudson County component and of the American Medical Association, died on August 29, 1985, at Riverside General Hospital, Secaucus, at the age of 82. A native of Cuba, Dr. Gonzalez was graduated from the Faculty of Medicine, Havana, Cuba, in 1926. He emigrated to the United States in 1962 and established a practice in general medicine in Union City. In 1976, Dr. Gonzalez was a recipient of MSNJ's Golden Merit Award, honoring him for his 50 years in medicine. He is survived by a son and daughter-in-law, Mario D. and Clara H. Gonzalez, both of whom are practicing physicians.

Dr. William V. Harz

A well-known surgeon in Bayonne, prior to his retirement, William Vincent Harz, M.D., died on August 27, 1985, at the age of 76. A native of Bayonne, Dr. Harz was graduated from Georgetown University School of Medicine in 1933. He was a member of our Hudson County component and of the American Medical Association, and was a Fellow of the International College of Surgeons. During World War II, Dr. Harz served in the medical department of the United States Air Force. In 1983, MSNJ conferred its Golden Merit Award on Dr. Harz in recognition of his 50 years as a physician.

Dr. Paul A. Kennedy

Paul Augustin Kennedy, M.D., former chief of medicine and chief of cardiology at Holy Name Hospital, Teaneck, died on September 9, 1985, in Orlando, Florida, at the venerable age of 84. Born in Union City, Dr. Kennedy was graduated from Columbia University's College of Physicians and Surgeons, New York, in 1927. He was a member of our Bergen County component, and of the American Medical Association. He had been a Fellow of the American College of Physicians, and a Diplomate of the American Board of Internal Medicine and of the American Board of Cardiologists.

Dr. William A. Lee

William Allen Lee, M.D., former chief of staff at Meadowview Hospital, Secaucus, for 30 years, died on September 8, 1985, at Christ Hospital, Jersey City, at the age of 73. Born in Brooklyn, Dr. Lee was graduated from the Eclectic Medical College, Cincinnati, Ohio, in 1938. A psychiatrist, Dr. Lee had a practice in Jersey City, and was affiliated with Christ Hospital and the Medical Center, Jersey City. He was a member of our Hudson County component and of the American Medical Association. Dr. Lee was a Fellow of the American Psychiatric Association. From 1942 to 1946, Dr. Lee served in the medical department of the United States Army, emerging with the rank of captain.

Dr. Carlos M. Lopez

At the untimely age of 57, Carlos Mario Lopez, M.D., a psychiatrist at Marlboro Psychiatric Hospital, died on June 10, 1985. Born in Campeche, Mexico, Dr. Lopez received his medical degree from the National University of Mexico at Merida, Yucatan, in 1956. He emigrated to the United States in 1957, serving his internship in Michigan and Maryland, and coming to the state of New Jersey in 1959. Dr. Lopez had been a member of our Monmouth County component and of the American Medical Association.

Dr. Benjamin Starr

A family practitioner, first in Jersey City and finally in West New York, Benjamin Starr, M.D., died on August 23, 1985, at the age of 73. Born in Russia, Dr. Starr was graduated from Eclectic Medical College, Cincinnati, Ohio, in 1939. He was a member of our Hudson County component and of the American Medical Association. Dr. Starr was a Fellow of the American Academy of Family Practice, and had an imposing list of hospital affiliations including St. Francis, Christ, and Fairmount Hospitals and the Medical Center, Jersey City, and North Hudson Hospital, Weehawken.

AUTHOR INFORMATION

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CONTENT

The educational content of each issue appears as scientific articles, based on research, original concepts relative to epidemiology of disease, and treatment methodology; case reports based on unusual clinical experiences; review articles; clinical notes, succinct items on some aspect or new observation or technique of a case experience; and special articles, which include evaluations, policy and position papers, and reviews of nonscientific subjects. Other topics include commentary (critical narration); medical history; therapeutic drug information; pediatric briefs; nutrition update; and an opinion column. Editorials are prepared by the Editor and by guest contributors on timely and relevant subjects; editorials are the responsibility of the author. The Doctors' Notebook section contains organizational, informational, and administrative items from MSNJ and from the community. Letters to the Editor and book reviews are welcome and will be published as space permits. The principal aim in the preparation of a contribution should be relevance to diagnosis and treatment and to education of patients and professionals. Preference will be given to professional authors from New Jersey and to out-of-state lecturers who submit a suitable manuscript based on a presentation made in New Jersey.

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1. Goldwyn RM: Subcutaneous mastectomy. *J Med Soc NJ* 74:1050-1052, 1977.
2. Dixon WJ, Massey FJ: *Introduction to Statistical Analysis.* New York, NY, McGraw-Hill, 1969, pp. 42-48.

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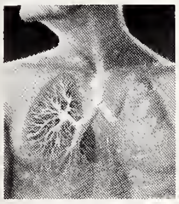

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BACTRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION. Clinical signs, such as rash, sore throat, fever, pallor, purpura or jaundice, may be early indications of serious reactions. In rare instances a skin rash may be followed by more severe reactions, such as Stevens-Johnson syndrome, toxic epidermal necrolysis, hepatic necrosis or serious blood disorder. Perform complete blood counts frequently.

BACTRIM SHOULD NOT BE USED IN THE TREATMENT OF STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have a greater incidence of bacteriologic failure when treated with Bactrim than with penicillin.

PRECAUTIONS:

General: Give with caution to patients with impaired renal or hepatic function, possible folate deficiency and severe allergy or bronchial asthma. In glucose-6-phosphate dehydrogenase-deficient individuals, hemolysis may occur, frequently dose-related.

Information for Patients: Instruct patients to maintain adequate fluid intake to prevent crystalluria and stone formation.

Laboratory Tests: Perform complete blood counts frequently; if a significant reduction in the count of any formed blood element is noted, discontinue Bactrim. Perform urinalyses with careful microscopic examination and renal function tests during therapy, particularly for patients with impaired renal function.

Drug Interactions: In elderly patients concurrently receiving certain diuretics, primarily thiazides, an increased incidence of thrombocytopenia with purpura has been reported. Bactrim may prolong the prothrombin time in patients who are receiving the anticoagulant warfarin. Keep this in mind when Bactrim is given to patients already on anticoagulant therapy and reassess coagulation time. Bactrim may inhibit the hepatic metabolism of phenytoin. Given at a common clinical dosage, it increased the phenytoin half-life by 39% and decreased the phenytoin metabolic clearance rate by 27%. When giving these drugs concurrently, be alert for possible excessive phenytoin effect. Sulfonamides can displace methotrexate from plasma protein binding sites, thus increasing free methotrexate concentrations.

Drug/Laboratory Test Interactions: Bactrim, specifically the trimethoprim component, can interfere with a serum methotrexate assay as determined by the competitive binding protein technique (CBPA) when a bacterial dihydrofolate reductase is used as the binding protein. No interference occurs if methotrexate is measured by a radioimmunoassay (RIA). The presence of trimethoprim and sulfamethoxazole may also interfere with the Jaffé alkaline picrate reaction assay for creatinine, resulting in overestimations of about 10% in the range of normal values.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Carcinogenesis: Long-term studies in animals to evaluate carcinogenic potential not conducted with Bactrim. *Mutagenesis:* Bacterial mutagenic studies not performed with sulfamethoxazole and trimethoprim in combination. Trimethoprim demonstrated to be nonmutagenic in the Ames assay. No chromosomal damage observed in human leukocytes *in vitro* with sulfamethoxazole and trimethoprim alone or in combination; concentrations used exceeded blood levels of these compounds following therapy with Bactrim. Observations of leukocytes obtained from patients treated with Bactrim revealed no chromosomal abnormalities. *Impairment of Fertility:* No adverse effects on fertility or general reproductive performance observed in rats given oral dosages as high as 70 mg/kg/day trimethoprim plus 350 mg/kg/day sulfamethoxazole.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Trimethoprim and sulfamethoxazole may interfere with folic acid metabolism; use during pregnancy only if potential benefit justifies potential risk to fetus. Nonteratogenic Effects: See CONTRAINDICATIONS section.

Nursing Mothers: See CONTRAINDICATIONS section.

Pediatric Use: Not recommended for infants under two months (see INDICATIONS and CONTRAINDICATIONS sections).

ADVERSE REACTIONS: Most common are gastrointestinal disturbances (nausea, vomiting, anorexia) and allergic skin reactions (such as rash and urticaria). **FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS (SEE WARNINGS SECTION).** *Hematologic:* Agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, neutropenia, hemolytic anemia, megaloblastic anemia, hypoprothrombinemia, methemoglobinemia, eosinophilia. *Allergic Reactions:* Stevens-Johnson syndrome, toxic epidermal necrolysis, anaphylaxis, allergic myocarditis, erythema multiforme, exfoliative dermatitis, angioedema, drug fever, chills, Henoch-Schoenlein purpura, serum sickness-like syndrome, generalized allergic reactions, generalized skin eruptions, photosensitivity, conjunctival and scleral injection, pruritus, urticaria and rash. Periarteritis nodosa and systemic lupus erythematosus have been reported. *Gastrointestinal:* Hepatitis (including cholestatic jaundice and hepatic necrosis), elevation of serum transaminase and bilirubin, pseudomembranous enterocolitis, pancreatitis, stomatitis, glossitis, nausea, emesis, abdominal pain, diarrhea, anorexia. *Genitourinary:* Renal failure, interstitial nephritis, BUN and serum creatinine elevation, toxic nephrosis with oliguria and anuria, crystalluria. *Neurologic:* Aseptic meningitis, convulsions, peripheral neuritis, ataxia, vertigo, tinnitus, headache. *Psychiatric:* Hallucinations, depression, apathy, nervousness. *Endocrine:* Sulfonamides bear certain chemical similarities to some goitrogens, diuretics (acetazolamide and the thiazides) and oral hypoglycemic agents; cross-sensitivity may exist. Diuresis and hypoglycemia have occurred rarely in patients receiving sulfonamides. *Musculoskeletal:* Arthralgia, myalgia. *Miscellaneous:* Weakness, fatigue, insomnia.

DOSAGE AND ADMINISTRATION: Not recommended for use in infants less than two months of age.

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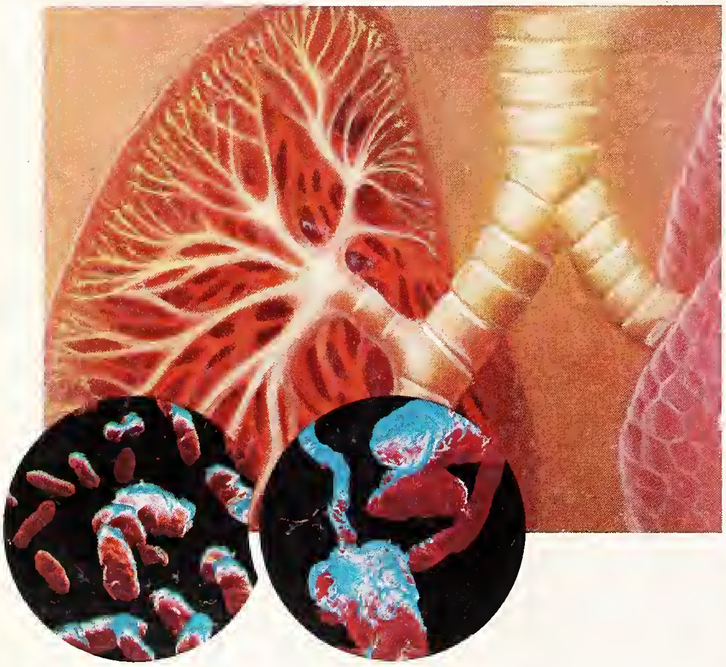

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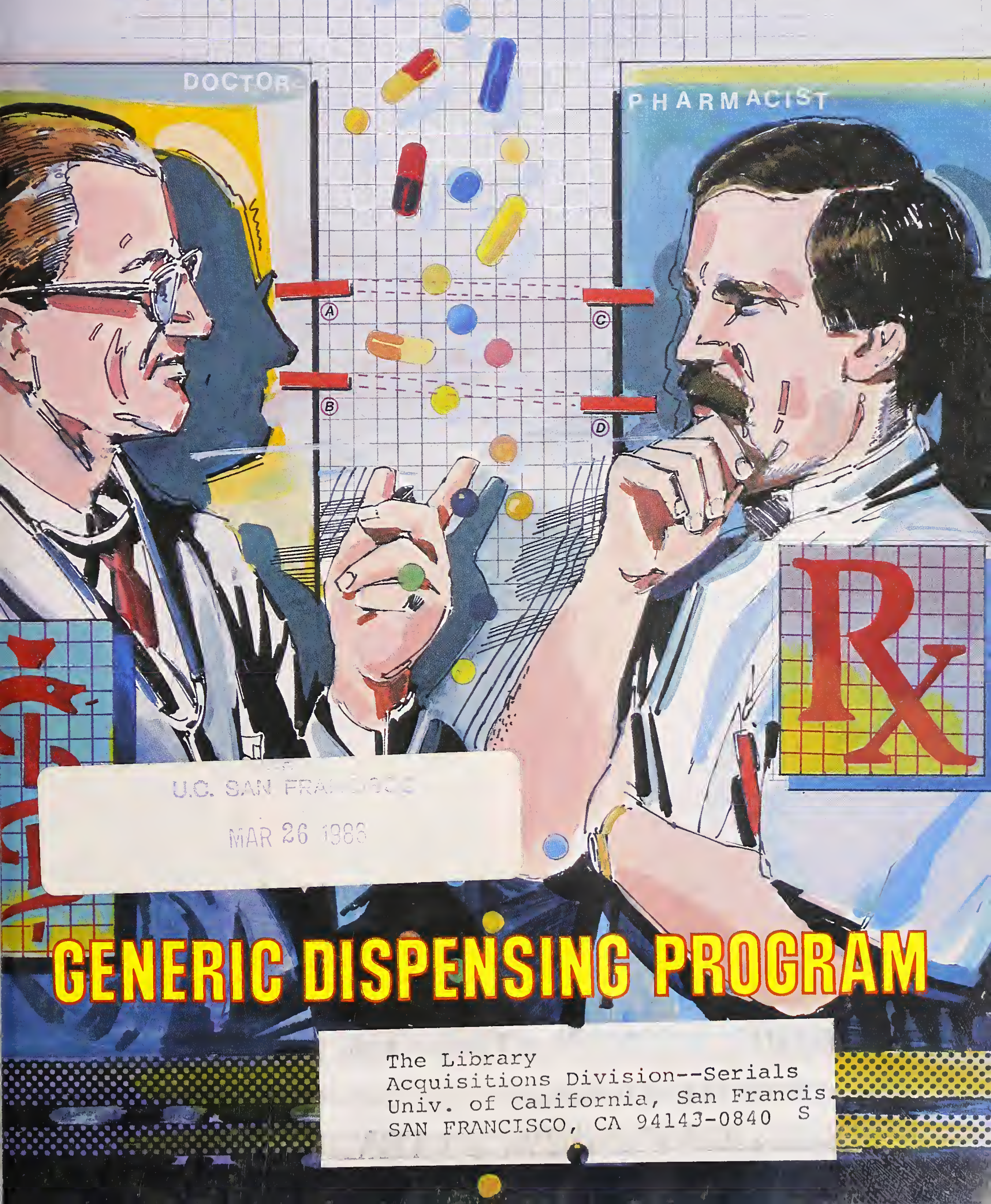

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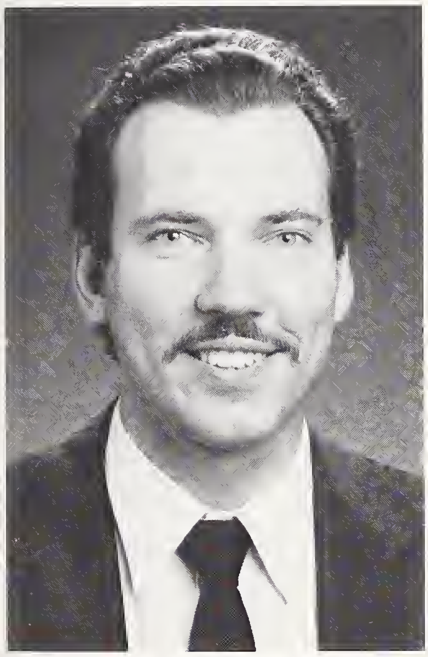

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On The Cover: New Jersey physicians and pharmacists were surveyed concerning a dual copay prescription program. Read the results, beginning on page 153. Cover illustration by William Harmuth.

New
WELLBUTRIN®
(Bupropion HCl)
75 mg Tablets
HELPING PATIENTS
REDISCOVER THE BEAUTY OF LIFE
Please see final pages for summary of prescribing information.
Wellcome

New

WELLBUTRIN®

(Bupropion HCl)

75 mg Tablets

an antidepressant of the aminoketone class, chemically unrelated to tricyclic, tetracyclic, or other known antidepressant agents

FOR THE TREATMENT OF DEPRESSION

- *In patients who fail to respond adequately to other antidepressant treatments.*
- *In patients who cannot tolerate alternative antidepressant treatments.*

Wellbutrin may be of particular benefit in patients who have experienced orthostatic hypotension, daytime drowsiness, or excessive weight gain while on other antidepressants.

---

*Wellbutrin is *not* recommended as an antidepressant of first choice for most patients because its use at doses approximately one and one-third times greater than the usually required daily dose (450 mg) is associated with a high risk of seizure (see WARNINGS in summary of prescribing information).

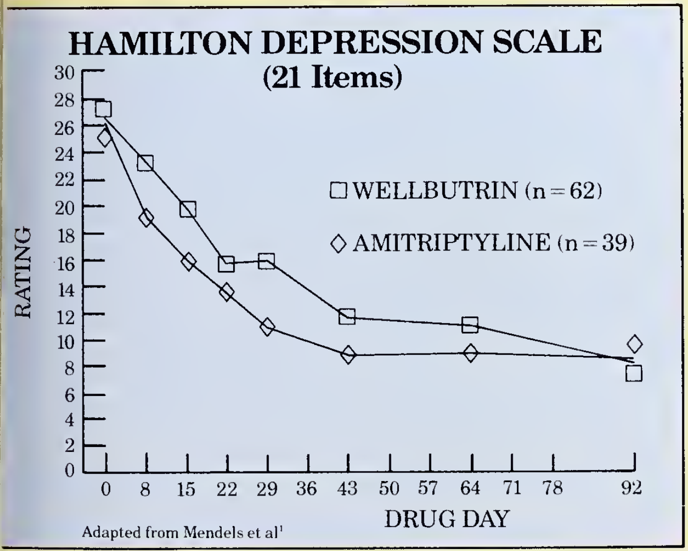
New **WELLBUTRIN®** (Bupropion HCl)

HELPING PATIENTS *who cannot tolerate the cardiovascular effects of alternative treatments*

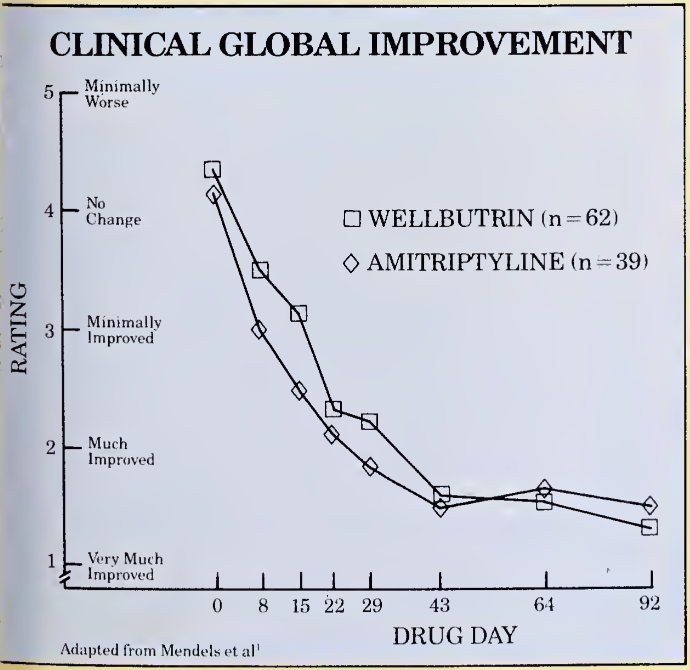
EFFICACY COMPARABLE TO AMITRIPTYLINE

hile many patients have been treated with ellbutrin in long-term clinical trials of up to two ears duration, there has been no systematic placebo-ntrolled evaluation of the efficacy of Wellbutrin for a eriod beyond three to four weeks.

double-blind controlled trial comparing Wellbutrin to mitriptyline was conducted at six centers.[1] No significant difference was found in therapeutic response to the vo drugs.



Adapted from Mendels et al[1]

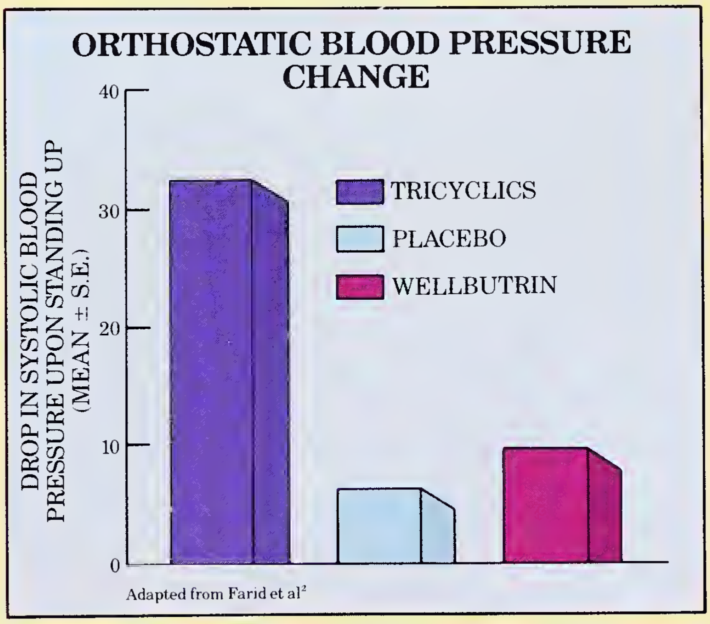


Adapted from Mendels et al[1]

Double-blind comparison of Wellbutrin and amitriptyline. Dosages were 300-450 ng/day for Wellbutrin, 75-150 mg/day for amitriptyline.

REDUCED INCIDENCE OF ORTHOSTATIC HYPOTENSION

In a study of twelve patients who had previously exhibited significant orthostatic hypotension while on tricyclic or tetracyclic medications, not one displayed clinically significant orthostatic hypotension after being switched to Wellbutrin.[2]



Adapted from Farid et al[2]

NO SIGNIFICANT EFFECT ON HEART RATE OR CARDIAC CONDUCTION

Wellbutrin has no more effect on heart rate or cardiac conduction than placebo. In a double-blind study with 23 inpatients comparing Wellbutrin (300-750 mg/day) to amitriptyline (75-225 mg/day), Wellbutrin produced no significant changes in any of the ECG parameters measured. By contrast, amitriptyline caused a significant prolongation in PR interval and QRS duration, as well as a decrease in QRS height.[3] In placebo-controlled clinical trials, the incidence of tachycardia on Wellbutrin was 10.8% vs. 8.6% on placebo.

There is no clinical experience establishing the safety of Wellbutrin in patients with a recent history of myocardial infarction or unstable heart disease. Care should be exercised if Wellbutrin is used in these groups.

Please see final pages for summary of prescribing information.

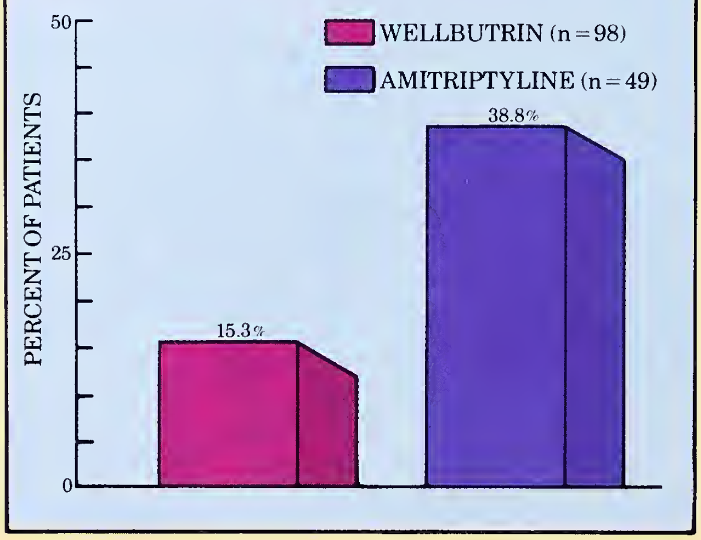
New WELLBUTRIN® (Bupropion HCl)

HELPING PATIENTS *who cannot tolerate the daytime drowsiness or weight gain associated with alternative treatments*

LOWER INCIDENCE OF DAYTIME DROWSINESS

In studies with 147 depressed patients, Wellbutrin caused substantially less drowsiness than amitriptyline.

PERCENT OF PATIENTS REPORTING TREATMENT-RELATED DROWSINESS



Without daytime drowsiness, patients may be better able to function during normal daily activities. Due to its relatively low incidence of daytime drowsiness, Wellbutrin may be advantageous for patients who need to drive a car or operate machinery. In placebo-controlled clinical trials, the incidence of dizziness on Wellbutrin was 22% vs. 16% on placebo. THEREFORE, CAUTION SHOULD BE EXERCISED UNTIL REASONABLY CERTAIN THE DRUG WILL NOT AFFECT PERFORMANCE.

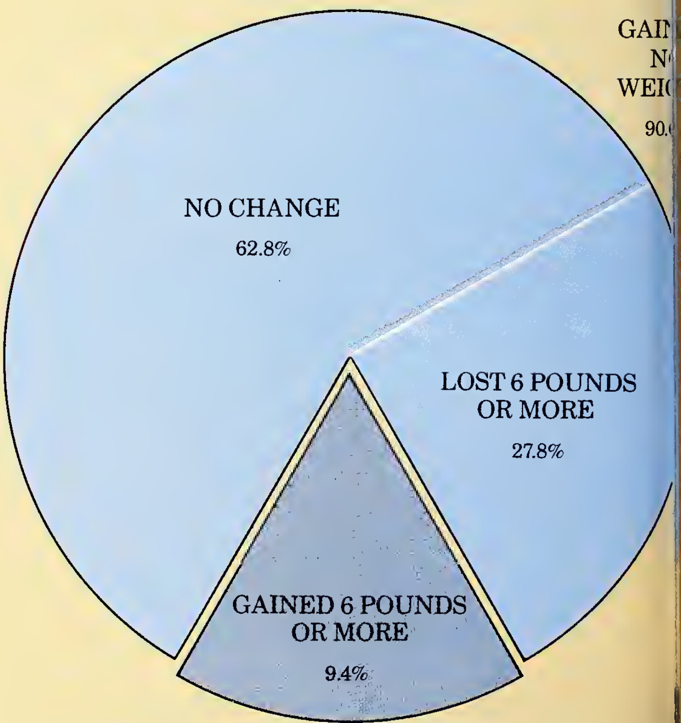
Some patients may report agitation or restlessness during the initial phase of therapy. In depressed patients with severe insomnia or pronounced agitation and restlessness, concurrent administration of a hypnotic during the first week of treatment may be helpful. For most patients, as the depression is brought under control, normal sleep patterns should return and the hypnotic may be removed.

In approximately 2% of patients in clinical trials, symptoms of restlessness, agitation, anxiety, and insomnia were sufficiently severe to require discontinuation of treatment with Wellbutrin.

LOW INCIDENCE OF WEIGHT GAIN

Long or short-term administration of Wellbutrin l
not been associated with significant weight gain. T
average patient should show no clinically signific
weight change during treatment.

CHANGE IN BODY WEIGHT
(% of Patients)

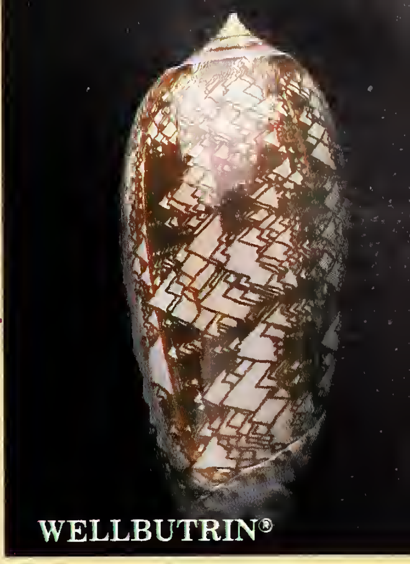


In active and placebo-controlled trials, involving 3
Wellbutrin patients, 90.6% experienced no weight ga
(± 5 pounds was considered normal weight fluctu
tion). 27.8% of patients lost six pounds or more. Th
possible effect should be considered when prescribi
for patients who have lost excessive weight in assoc
tion with their depression. In placebo-controlled cli
cal trials, the incidence of nausea/vomiting
Wellbutrin was 23% vs. 19% on placebo.

Furthermore, 34.5% of patients receiving tricyclic an
depressants in clinical trials gained weight, in contra
to only 9.4% of patients treated with Wellbutrin.

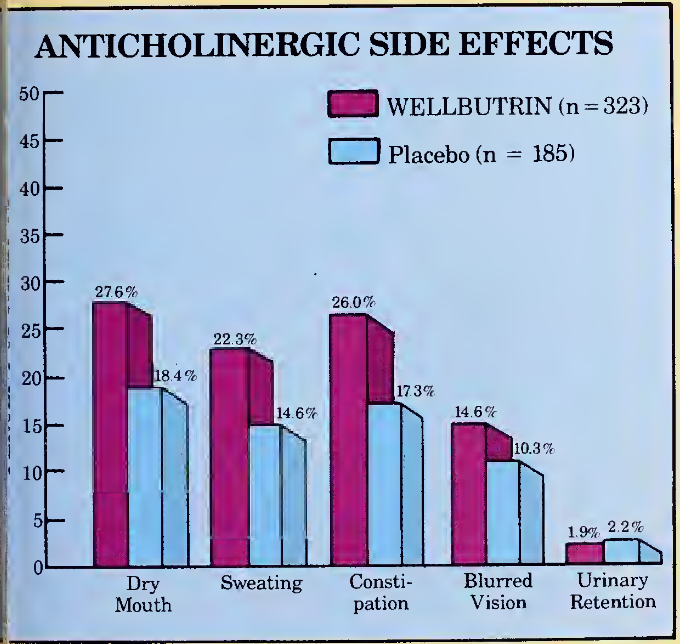
New WELLBUTRIN® (Bupropion HCl)

HELPING PATIENTS *who cannot tolerate* *e anticholinergic side effects of alternative treatments*



PROFILE OF SELECTED ANTICHOLINERGIC SIDE EFFECTS

ticholinergic side effects are weak or absent in most ses. Mild and transient dry mouth, the most frequent llbutrin anticholinergic side effect, was reported in 6% of patients.


ANTICHOLINERGIC SIDE EFFECTS

SEIZURES

At doses approximately one and one-third times greater than the usually required daily dose (450 mg), Wellbutrin is associated with a high risk of seizures. The estimated risk increases almost tenfold between a dose of 450 and 600 mg a day. Given the wide variability among individuals in their capacity to metabolize and eliminate drugs, this disproportional increase in seizure incidence with dose incrementation is a cause for concern. The estimated risk of seizures at doses of 450 mg and below does not appear excessive in comparison to the risk reported for other antidepressant drug products.

Seizures are a well-recognized consequence of tricyclic antidepressant therapy, and have been reported to occur in normal clinical use at a frequency of approximately 0.1 to 1.0%.[4,5]

INCIDENCE OF SEIZURES IN PATIENTS RECEIVING WELLBUTRIN

	WELLBUTRIN Dose mg/day	Total Seizure Incidence (%)	Seizure Incidence in Patients Without Seizure Predisposition (%)
within the recommended dose	less than 450	0.2%	0.0%
	450	0.3%	0.2%
above the recommended dose	600	2.3%	1.3%
	600-900	2.8%	1.9%

The risk of seizures with Wellbutrin appears to be strongly associated with dose and may be increased by predisposing factors (e.g., head trauma, CNS tumor, etc.) or a history of prior seizure. Extreme caution should be used when administering Wellbutrin to these patients or when combining Wellbutrin with other agents which lower seizure threshold (see WARNINGS in summary of prescribing information).

Please see final pages for summary of prescribing information.

New WELLBUTRIN® (Bupropion HCl)

HELPING PATIENTS REDISCOVER THE BEAUTY OF LIFE

- *Cautious initial usage recommended*
- *Doses above 450 mg/day should not be used*

RECOMMENDED DOSING

Wellbutrin is available in 75 mg tablets, and should be administered three times a day (T.I.D.) to help avoid high peak concentrations. Peak plasma concentrations are usually achieved within 2 hours, followed by a biphasic decline. The average half-life of the second phase is approximately 14 hours, with a range of 8 to 24 hours.

The recommended starting dose is 225 mg/day, with a gradual ascension up to the maximum recommended dose of 450 mg/day based upon the clinical response of the patient. Dosage may be increased by 75 mg/day no sooner than every three days, and, as shown below, the maximum number of tablets for a single dose is two.

DOSING SCHEDULE

Treatment Day	75 mg Tablets Morning	 Midday	 Evening	Total Daily Dose
1	1	1	1	225 mg
4	2	1	1	300 mg
7	2	2	1	375 mg
10	2	2	2	450 mg

In general, older patients are known to metabolize drugs more slowly and to be more sensitive to the anticholinergic, sedative, and cardiovascular side effects of antidepressant drugs. Clinical trials enrolled several hundred patients 60 years of age and older. The experience with these patients and younger ones was similar.

Treatment of patients with renal or hepatic impairment should be initiated at reduced dosage as Wellbutrin and its metabolites may accumulate in such patients beyond concentrations expected in patients without renal or hepatic impairment. These patients should be closely monitored for possible toxic effects of elevated blood and tissue levels of drug and metabolites.

The lowest dose which maintains remission is recommended. If patients do not demonstrate an adequate response after an appropriate period of treatment at 450 mg/day, the product should be discontinued (see WARNINGS in summary of prescribing information).

Adverse events were sufficiently troublesome to cause discontinuation of Wellbutrin treatment in approximately ten percent of the 2400 patients and volunteers who participated in the product's premarketing clinical trials.

OVERDOSE EXPERIENCE

There has been limited clinical experience with ov
dosage of Wellbutrin. Thirteen overdoses occurred d
ing clinical trials. Twelve patients ingested 850 to 42
mg and recovered without significant sequel
Another patient who ingested 9000 mg of Wellbut
and 300 mg of tranylcypromine experienced a gra
mal seizure and recovered without further sequel
None of these thirteen patients displayed clinically s
nificant cardiovascular abnormalities. As with a
antidepressant, care should be taken to lessen the ri
of suicide by writing prescriptions for the smalle
number of tablets consistent with good patient ma
agement.

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1. Mendels J, Amin MM, Chouinard G, et al: A comparative study of bupropion
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4. Lowry MR, Dunner FJ: Seizures during tricyclic therapy. *Am J Psychiatry* 1980; 137
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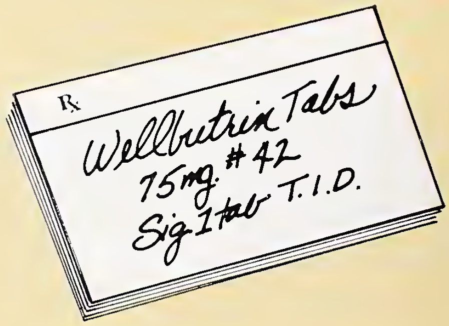

New WELLBUTRIN®

(Bupropion HCl)

HELPING PATIENTS REDISCOVER THE BEAUTY OF LIFE

LLBUTRIN® (BUPROPION HYDROCHLORIDE) Tablets.

ummary:

IONS AND USAGE:
itrin is indicated for the treatment of depression in patients who fail to respond adequately to or
iot tolerate alternative antidepressant treatments. Wellbutrin is *not* recommended as an antide-
of first choice for most patients because its use at doses approximately one and one-third times
han the usually required daily dose (450 mg) is associated with a high risk of seizure (see WARN-

ficacy of Wellbutrin was demonstrated in placebo controlled clinical trials which enrolled princi-
pitalized patients with diagnoses of depressive neurosis or manic-depressive (depressed type)
The depressive illness of the patients studied most closely corresponds to the Major Depression
of the APA Diagnostic and Statistical Manual III.
Depression implies a prominent and relatively persistent depressed or dysphoric mood that
iterferes with daily functioning (nearly every day for at least two weeks); it should include at least
he following eight symptoms: change in appetite, change in sleep, psychomotor agitation or
on, loss of interest in usual activities or decrease in sexual drive, increased fatigability, feelings of
orthlessness, slowed thinking or impaired concentration, and suicidal ideation or attempts.
ice to demonstrate the sustained effectiveness of Wellbutrin after three weeks of use in pla-
itrolled investigations is not presently available.

AINDICATIONS:
ise of its potential to induce seizures, Wellbutrin should not be used in patients with a convulsive

oncurrent administration of Wellbutrin and a monoamine oxidase (MAO) inhibitor is contraindi-
least 14 days should elapse between discontinuation of an MAO inhibitor and initiation of treat-
h Wellbutrin.
itrin is contraindicated in patients who have shown an allergic response to it.

IGS:

LSIONS:
**utrin appears to possess a greater epileptogenic potential than other marketed antide-
its. While the estimated risk of seizure at doses of 450 mg and below does not appear
ve in comparison to the risk reported for other antidepressant drug products, the esti-
isk increases almost tenfold between a dose of 450 and 600 mg a day. Given the wide
ity among individuals in their capacity to metabolize and eliminate drugs, this dispro-
ate increase in seizure incidence with dose incrementation is a cause for concern.**
**ig the period of premarketing evaluation, 25 among approximately 2400 patients
with Wellbutrin experienced seizures. At the time of seizure, seven (7) patients were
ig daily doses of Wellbutrin at or below the lowest documented effective daily dose of
. Twelve (12) patients experienced seizures at daily doses of 600 mg; six (6) additional
had seizures at daily doses between 600 and 900 mg. The risk of seizure appears to be
associated with dose and may be increased by predisposing factors (e.g., head trauma,
nor, etc.) or a history of prior seizure. In addition, sudden and large increments in dose
itribute to an increased risk. While many seizures occurred early in the course of treat-
ome seizures did occur after several weeks of use at fixed dose.**

INCIDENCE OF SEIZURES IN PATIENTS RECEIVING WELLBUTRIN

	Wellbutrin Dose mg/day	Total Seizure Incidence (%)	Seizure Incidence in Patients Without Seizure Predisposition (%)
ithin nmended)ose	<450	0.2%	0.0%
	450	0.3%	0.2%
bove nmended)ose	600	2.3%	1.3%
	600-900	2.8%	1.9%

**IGE AND ADMINISTRATION RECOMMENDATIONS SHOULD BE STRICTLY FOLLOWED TO
ZE THE RISK OF SEIZURE (see DOSAGE AND ADMINISTRATION).**
**e caution should be used when combining Wellbutrin with other agents which lower sei-
eshold or when administering Wellbutrin to patients with a history of seizure disorder or
trauma.**
al for Hepatotoxicity: In rats receiving large doses of bupropion chronically, there was an
in incidence of hepatic hyperplastic nodules and hepatocellular hypertrophy. In dogs receiving
ses of bupropion chronically, various histologic changes were seen in the liver, and laboratory
ggesting mild hepatocellular injury were noted. Although scattered abnormalities in liver function
re detected in patients participating in clinical trials, there is no clinical evidence that bupropion
hepatotoxin in humans.

JTIONS:

I:
tion and Insomnia: A substantial proportion of patients treated with Wellbutrin experience
egree of increased restlessness, agitation, anxiety, and insomnia, especially shortly after initiation
ment. In clinical studies, these symptoms were sometimes of sufficient magnitude to require
nt with sedative/hypnotic drugs. In approximately 2% of patients, symptoms were sufficiently
o require discontinuation of Wellbutrin treatment.
hosis, Confusion, and Other Neuropsychiatric Phenomena: Patients treated with Wellbutrin
en reported to show a variety of neuropsychiatric signs and symptoms including delusions, hallu-
s, psychotic episodes, confusion, and paranoia. Because of the uncontrolled nature of many stud-
impossible to provide a precise estimate of the extent of risk imposed by treatment with
in. In several cases, neuropsychiatric phenomena abated upon dose reduction and/or with-
f treatment.
ation of Psychosis and/or Mania: Antidepressants can precipitate manic episodes in Bipolar
epressive patients during the depressed phase of their illness and may activate latent psychosis
susceptible patients. Wellbutrin is expected to pose similar risks.
ed Appetite and Weight: A weight loss of greater than 5 pounds occurred in 28% of Wellbutrin
. This incidence is approximately double that seen in comparable patients treated with tricyclics
bo. Furthermore, while 34.5% of patients receiving tricyclic antidepressants gained weight, only
patients treated with Wellbutrin did. Consequently, if weight loss is a major presenting sign of a
depressive illness, the anorectic and/or weight reducing potential of Wellbutrin should be con-

de: The possibility of a suicide attempt is inherent in depression and may persist until significant
in occurs. Accordingly, prescriptions for Wellbutrin should be written for the smallest number of
consistent with good patient management.
Patients with Systemic Illness: There is no clinical experience establishing the safety of
in in patients with a recent history of myocardial infarction or unstable heart disease. Therefore,
uld be exercised if it is used in these groups. Wellbutrin was well tolerated in patients who had
sly developed orthostatic hypotension while receiving tricyclic antidepressants.
use bupropion HCl and its metabolites are almost completely excreted through the kidney and
lites are likely to undergo conjugation in the liver prior to urinary excretion, treatment of patients
al or hepatic impairment should be initiated at reduced dosage as bupropion and its metabolites
cumulate in such patients beyond concentrations expected in patients without renal or hepatic
ient. The patient should be closely monitored for possible toxic effects of elevated blood and
vels of drug and metabolites.
ation for Patients: Physicians are advised to discuss the following issues with patients:
nts should be instructed to take Wellbutrin in equally divided doses three or four times a day to minimize the risk of seizure.

Patients should be told that any CNS-active drug like Wellbutrin may impair their ability to perform tasks requiring judgment or motor and cognitive skills. Consequently, until they are reasonably certain that Wellbutrin does not adversely affect their performance they should refrain from driving an automobile or operating complex, hazardous machinery.

Patients should be told that the use and cessation of use of alcohol may alter the seizure threshold, and, therefore, that the consumption of alcohol should be minimized, and, if possible, avoided completely.

Patients should be advised to inform their physician if they are taking or plan to take any prescription or over-the-counter drugs. Concern is warranted because Wellbutrin and other drugs may affect each other's metabolism.

Patients should be advised to notify their physician if they become pregnant or intend to become pregnant during therapy.

Drug Interactions: No systematic data have been collected on the consequences of the concomitant administration of Wellbutrin and other drugs.

However, animal data suggest that Wellbutrin may be an inducer of drug metabolizing enzymes. This may be of potential clinical importance because the blood levels of co-administered drugs may be altered.

Alternatively, because bupropion is extensively metabolized, the co-administration of other drugs may affect its clinical activity. In particular, care should be exercised when administering drugs known to affect hepatic drug metabolizing enzyme systems (e.g., carbamazepine, cimetidine, phenobarbital, phenytoin).

Studies in animals demonstrate that the acute toxicity of bupropion is enhanced by the MAO inhibitor phenelzine (see CONTRAINDICATIONS).

Limited clinical data suggest a higher incidence of adverse experiences in patients receiving concurrent administration of Wellbutrin and L-dopa. Administration of Wellbutrin to patients receiving L-dopa concurrently should be undertaken with caution, using small initial doses and small gradual dose increases.

Concurrent administration of Wellbutrin and agents which lower seizure threshold should be undertaken only with extreme caution (see WARNINGS). Low initial dosing and small gradual dose increases should be employed.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Lifetime carcinogenicity studies were performed in rats and mice at doses up to 300 and 150 mg/kg/day, respectively. In the rat study there was an increase in nodular proliferative lesions of the liver at doses of 100 to 300 mg/kg/day; lower doses were not tested. The question of whether or not such lesions may be precursors of neoplasms of the liver is currently unresolved. Similar liver lesions were not seen in the mouse study, and no increase in malignant tumors of the liver and other organs was seen in either study.

Bupropion produced a borderline positive response (2-3 times control mutation rate) in some strains in the Ames bacterial mutagenicity test, and a high oral dose (300, but not 100 or 200 mg/kg) produced a low incidence of chromosomal aberrations in rats. The relevance of these results in estimating the risk of human exposure to therapeutic doses is unknown.

A fertility study was performed in rats; no evidence of impairment of fertility was encountered at oral doses up to 300 mg/kg/day.

Pregnancy: *Teratogenic Effects:* Pregnancy Category B: Reproduction studies have been performed in rabbits and rats at doses up to 15-45 times the human daily dose and have revealed no definitive evidence of impaired fertility or harm to the fetus due to bupropion. (In rabbits, a slightly increased incidence of fetal abnormalities was seen in two studies, but there was no increase in any specific abnormality). There are no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Labor and Delivery: The effect of Wellbutrin on labor and delivery in humans is unknown.

Nursing Mothers: It is not known whether Wellbutrin is excreted in the milk of lactating women. Because many drugs are excreted in human milk, caution should be exercised when Wellbutrin is administered to women who are nursing.

Pediatric Use: The safety and effectiveness of Wellbutrin in individuals under 18 years old have not been established.

Use in the Elderly: Wellbutrin has not been systematically evaluated in older patients.

ADVERSE REACTIONS: (See also WARNINGS and PRECAUTIONS) Adverse events commonly encountered in patients treated with Wellbutrin are agitation, dry mouth, insomnia, headache/migraine, nausea/vomiting, constipation, and tremor.

Adverse events were sufficiently troublesome to cause discontinuation of Wellbutrin treatment in approximately ten percent of the 2400 patients and volunteers who participated in the product's premarketing clinical trials. The more common events causing discontinuation include neuropsychiatric disturbances (3.0%), primarily agitation and abnormalities in mental status; gastrointestinal disturbances (2.1%), primarily nausea and vomiting; neurological disturbances (1.7%), primarily seizures, headaches, and sleep disturbances; and dermatologic problems (1.4%), primarily rashes. It is important to note, however, that many of these events occurred at doses that exceed the recommended daily dose.

Accurate estimates of the incidence of adverse events associated with the use of any drug are difficult to obtain. Estimates are influenced by drug dose, detection technique, setting, physician judgments, etc. Consequently, the table below is presented solely to indicate the relative frequency of adverse events reported in representative controlled clinical studies conducted to evaluate the safety and efficacy of Wellbutrin under relatively similar conditions of daily dosage (300-600mg), setting, and duration (3-4 weeks). The figures cited cannot be used to predict precisely the incidence of untoward events in the course of usual medical practice where patient characteristics and other factors must differ from those which prevailed in the clinical trials. These incidence figures also cannot be compared with those obtained from other clinical studies involving related drug products as each group of drug trials is conducted under a different set of conditions.

Finally, it is important to emphasize that the tabulation does not reflect the relative severity and/or clinical importance of the events. A better perspective on the serious adverse events associated with the use of Wellbutrin is provided in the WARNINGS and PRECAUTIONS sections.

TREATMENT EMERGENT ADVERSE EXPERIENCE INCIDENCE IN PLACEBO-CONTROLLED CLINICAL TRIALS*
(Percent of Patients Reporting)

Adverse Experience	Wellbutrin Patients (n = 323)	Placebo Patients (n = 185)
CARDIOVASCULAR		
CARDIAC ARRHYTHMIAS	5.3	4.3
DIZZINESS	22.3	16.2
HYPERTENSION	4.3	1.6
HYPOTENSION	2.5	2.2
PALPITATIONS	3.7	2.2
SYNCOPE	1.2	0.5
TACHYCARDIA	10.8	8.6
DERMATOLOGIC		
PRURITIS	2.2	0.0
RASH	8.0	6.5
GASTROINTESTINAL		
ANOREXIA	18.3	18.4
APPETITE INCREASE	3.7	2.2
CONSTIPATION	26.0	17.3
DIARRHEA	6.8	8.6

Adverse Experience	Wellbutrin Patients (n = 323)	Placebo Patients (n = 185)
GASTROINTESTINAL		
DYSPEPSIA	3.1	2.2
NAUSEA/VOMITING	22.9	18.9
WEIGHT GAIN	13.6	22.7
WEIGHT LOSS	23.2	23.2
GENITOURINARY		
IMPOTENCE	3.4	3.1
MENSTRUAL COMPLAINTS	4.7	1.1
URINARY FREQUENCY	2.5	2.2
URINARY RETENTION	1.9	2.2
MUSCULOSKELETAL		
ARTHRITIS	3.1	2.7
NEUROLOGICAL		
AKATHISIA	1.5	1.1
AKINESIA/BRADYKINESIA	8.0	8.6
CUTANEOUS TEMP DISTURBANCE	1.9	1.6
DRY MOUTH	27.6	18.4
EXCESSIVE SWEATING	22.3	14.6
HEADACHE/MIGRAINE	25.7	22.2
IMPAIRED SLEEP QUALITY	4.0	1.6
INCREASED SALIVARY FLOW	3.4	3.8
INSOMNIA	18.6	15.7
MUSCLE SPASMS	1.9	3.2
PSEUDOPARKINSONISM	1.5	1.6
SEDATION	19.8	19.5
SENSORY DISTURBANCE	4.0	3.2
TREMOR	21.1	7.6
NEUROPSYCHIATRIC		
AGITATION	31.9	22.2
ANXIETY	3.1	1.1
CONFUSION	8.4	4.9
DECREASED LIBIDO	3.1	1.6
DELUSIONS	1.2	1.1
DISTURBED CONCENTRATION	3.1	3.8
EUPHORIA	1.2	0.5
HOSTILITY	5.6	3.8
NONSPECIFIC		
FATIGUE	5.0	8.6
FEVER/CHILLS	1.2	0.5
RESPIRATORY		
UPPER RESP COMPLAINTS	5.0	11.4
SPECIAL SENSES		
AUDITORY DISTURBANCE	5.3	3.2
BLURRED VISION	14.6	10.3
GUSTATORY DISTURBANCE	3.1	1.1

[*] Events reported by at least 1% of Wellbutrin patients are included.

Other events observed during the entire premarketing *evaluation* of Wellbutrin: During its premarketing assessment, Wellbutrin was evaluated in almost 2400 subjects. The conditions and duration of exposure to Wellbutrin varied greatly and a substantial proportion of the experience was gained in open and uncontrolled clinical settings. During this experience, numerous adverse events were reported; however, without appropriate controls, it is impossible to determine with certainty which events were or were not caused by Wellbutrin. The following enumeration is organized by organ system and describes events in terms of their relative frequency of reporting in the data base. Events of major clinical importance are also described in the WARNINGS and PRECAUTIONS sections of the labeling.

The following definitions of frequency are used: Frequent adverse events are defined as those occurring in at least 1/100 patients. Infrequent adverse events are those occurring in 1/100 to 1/1000 patients, while rare events are those occurring in less than 1/1000 patients.

Cardiovascular: Frequent was edema; infrequent were chest pain, EKG abnormalities (premature beats and nonspecific ST-T changes), and shortness of breath/dyspnea; and rare were pallor and phlebitis.

Dermatologic: Frequent were nonspecific rashes; infrequent were alopecia and dry skin; rare were change in hair color and hirsutism.

Endocrine: Infrequent was gynecomastia; rare were glycosuria and hormone level change .

Gastrointestinal: Infrequent were dysphagia, thirst disturbance, and liver damage/jaundice; rare were rectal complaints, colitis, G.I. bleeding, and intestinal perforation.

Genitourinary: Frequent was nocturia; infrequent were vaginal irritation, testicular swelling, urinary tract infection, painful erection, and retarded ejaculation; rare were dysuria, enuresis, urinary incontinence, menopause, ovarian disorder, pelvic infection, cystitis, dyspareunia, and painful ejaculation.

Hematologic/Oncologic: Rare was lymphadenopathy.

Neurological: (see WARNINGS) Frequent were ataxia/incoordination, seizure, myoclonus, dyskinesia, and dystonia; infrequent were mydriasis, vertigo, and dysarthria; and rare were EEG abnormality, abnormal neurological exam, impaired attention, and sciatica.

Neuropsychiatric: (see PRECAUTIONS) Frequent were mania/hypomania, increased libido, hallucinations, decrease in sexual function, and depression; infrequent were memory impairment, depersonalization, psychosis, dysphoria, mood instability, paranoia, formal thought disorder, and frigidity; rare was suicidal ideation.

Oral Complaints: Frequent was stomatitis; infrequent were toothache, bruxism, gum irritation, and oral edema; rare was glossitis.

Respiratory: Infrequent were bronchitis and shortness of breath/dyspnea; rare were epistaxis and rate or rhythm disorder.

Special Senses: Infrequent was visual disturbance; rare was diplopia.

Nonspecific: Frequent were flu-like symptoms; infrequent was nonspecific pain; rare were body odor, surgically related pain, infection, medication reaction and overdose.

DRUG ABUSE AND DEPENDENCE:

Humans: Controlled clinical studies conducted in normal volunteers, in subjects with a history of multiple drug abuse, and in depressed patients showed some increase in motor activity and agitation/excitement.

In a population of individuals experienced with drugs of abuse, a single dose of 400 mg Wellbutrin produced mild amphetamine-like activity as compared to placebo on the morphine-benzedri
of the Addiction Research Center Index (ARCI), and a score intermediate between placebo a
mine on the Liking Scale of the ARCI. These scales measure general feelings of euphoria an
ability.

Findings in clinical trials, however, are not known to predict the abuse potential of drugs re
theless, evidence from single dose studies does suggest that the recommended daily dosa
pion when administered in divided doses is not likely to be especially reinforcing to amp
stimulant abusers. However, higher doses, which could not be tested because of the risk of se
be modestly attractive to those who abuse stimulant drugs.

Animals: Studies in rodents have shown that bupropion exhibits some pharmacologic actio
to psychostimulants, including increases in locomotor activity and the production of a mild
behavior and increases in rates of responding in several schedule-controlled behavior para
discrimination studies in rats showed stimulus generalization between bupropion and amphe
other psychostimulants. Rhesus monkeys have been shown to self-administer bupropion int

OVERDOSAGE:

Lethal doses in animals: In rats, the acute oral LD_{50} values were 607 mg/kg (males) and
(females). Respective values for mice were 544 mg/kg and 636 mg/kg. Signs of acute toxi
labored breathing, salivation, arched back, ptosis, ataxia, and convulsions.

Human overdose experience: There has been limited clinical experience with ov
Wellbutrin. Thirteen overdoses occurred during clinical trials. Twelve patients ingested 850
and recovered without significant sequelae. Another patient who ingested 9000 mg of Wellbu
mg of tranylcypromine experienced a grand mal seizure and recovered without further sequ

Management of *overdose*: Following suspected overdose, hospitalization is advised. If th
conscious, vomiting should be induced by syrup of ipecac. Activated charcoal also may be a
every 6 hours during the first 12 hours after ingestion. Baseline laboratory values should b
Electrocardiogram and EEG monitoring also are recommended for the next 48 hours. Ad
intake should be provided.

If the patient is stuporous, comatose, or convulsing, airway intubation is recommended pr
taking gastric lavage. Although there is little clinical experience with lavage following an
Wellbutrin, it is likely to be of benefit within the first 12 hours after ingestion since absorption
may not yet be complete.

While diuresis, dialysis, or hemoperfusion are sometimes used to treat drug overdosage,
experience with their use in the management of Wellbutrin overdose. Because diffusion o
from tissue to plasma may be slow, dialysis may be of minimal benefit several hours after ov

Based on studies in animals, it is recommended that seizures be treated with an intraven
diazepine preparation and other supportive measures, as appropriate.

Further information about the treatment of overdoses may be available from a poison con

DOSAGE AND ADMINISTRATION: At doses that are one and one-third times the usually re
(450 mg/day) (see WARNINGS), the observed incidence of seizure increases by as much as t
predicts a relatively narrow therapeutic ratio for the safe use of bupropion, especially as ther
erable inter-individual variability in the capacity of patients to metabolize drugs.

Consequently, it is particularly important to administer bupropion in a manner that is m
minimize the risk of seizure. Retrospective analysis of clinical experience gained during its pr
development suggests that the risk of seizure may be minimized if 1) the total daily dose o
does *not* exceed 450 mg, 2) the daily dose is administered in divided doses to avoid high pe
trations of bupropion and/or its metabolites, and 3) the rate of incrementation of dose is ver

Wellbutrin should be given t.i.d., preferably with intervals of at least 6 hours between succes
Besides its apparent value in lowering the risk of seizure, gradual dose escalation is important
motor restlessness, and insomnia, often seen during the initial days of treatment, are to be m
necessary, these side effects may be managed by temporary reduction of dose or the transito
tration of a sedative hypnotic. A sedative hypnotic usually is not required beyond the first we
ment.

The following suggested dosing regimen incorporates the principles described above.

Usual Adult Dosage: A starting dose of 225 mg/day given as 75 mg t.i.d. is recommended
clinical response, this dose may be increased by 75 mg/day no sooner than every three days a
the following schedule, up to a total maximum daily dose of 450 mg/day. Of course, if distre
ward effects supervene, dose escalation should be stopped.

DOSING SCHEDULE

Treatment Day	75 mg Tablets			Total Daily D
	Morning	Midday	Evening	
1	1	1	1	225 mg
4	2	1	1	300 mg
7	2	2	1	375 mg
10	2	2	2	450 mg

In patients who do not demonstrate an adequate response after an appropriate period of tre
450 mg/day, drug should be discontinued.

Elderly Patients: In general, older patients are known to metabolize drugs more slowly and t
sensitive to the anticholinergic, sedative, and cardiovascular side effects of antidepressant dru
trials enrolled several hundred patients 60 years of age and older. The experience with these pa
younger ones was similar.

Maintenance: The lowest dose which maintains remission is recommended. The maximu
mended maintenance dose is 450 mg/day. While it is generally recommended that a course
pressant drug treatment should continue for several months and many patients have been tre
Wellbutrin in long-term clinical trials of up to 2 years duration, there has been no systematic pla
trolled evaluation of the efficacy of Wellbutrin for a period beyond three to four weeks.

HOW SUPPLIED: Wellbutrin (bupropion hydrochloride) Tablets are supplied as 75 mg (yellow-go
biconvex tablets printed "WELLBUTRIN" and "75," bottles of 100 (NDC 0081-0177-55).
Store at 15°-25°C (59°-77°F).

[*] U.S. Patent No. 3819706
U.S. Patent No. 3885046 (Use Patent)

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MEMBERSHIP NEWSLETTER

MSNJ

THE MEDICAL SOCIETY OF NEW JERSEY VOLUME 31

MEDICARE FORMS

Physicians no longer can obtain complimentary supplies of the Health Care Financing Administration's Medicare claim forms from carriers. Standard form HCFA-1500, developed by the AMA, must be used for Medicare claims and, by October 1, 1986, will be required for Medicaid claims. Copies of the form are available from the AMA. The price for a carton of 1,000 single sheets is $34.10 (ask for OP-501). A carton of 1,000 two-part snapouts (OP-502) costs $49.50, and a two-part computer pinfeed (OP-503) costs $51.75. Prices include UPS and other charges. There is a discount for bulk orders. There is an additional 10 percent discount for AMA members and medical societies. Direct inquiries to AMA Insurance Form, Book and Pamphlet Fulfillment, AMA, P.O. Box 10946, Chicago, IL 60610-9968, or call (312) 280-7168. Allow two weeks for delivery.

MEDICARE BUDGET

Congress adjourned after being unable to complete the budget reconciliation process.

Before doing so, however, it took emergency action extending current Medicare provisions—a continuing freeze on fees for nonparticipating physicians and a freeze on reimbursement for participating physicians—until March 14. It marked the fourth time that Medicare and other provisions, originally scheduled to have expired at midnight on September 30, have been extended through emergency legislation.

Physicians should be advised that Medicare provisions remain unchanged from what they were before.

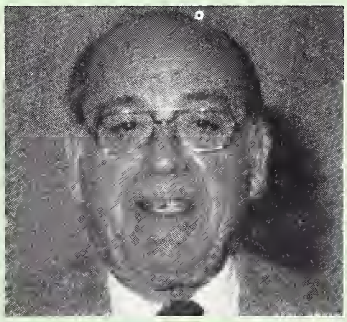
TOXIC SHOCK SYNDROME, BACTERIAL MENINGITIS, AND BACTEREMIA

The State Health Department and the Centers for Disease Control are establishing a special study for Toxic Shock Syndrome (TSS) in New Jersey. During the next 18 months all suspected cases of TSS, hospitalized or not, regardless of severity should be reported to the Health Department. The diagnosis should be considered in any patient with compatible clinical finding regardless of age or sex.

Bacterial meningitis and bacteremia also will be the subject of special studies. Particular emphasis will be paid to public health questions, including vaccine efficacy studies and disease control in child day-care settings.

The Medical Society of New Jersey urges all members to cooperate with the Health Department in these special studies. The project is under the supervision of David R. Cundiff, M.D., M.P.H., Assistant Director, Communicable Disease Control Services Office. If you have any questions related to project staff interviewers or any other issue regarding the project when you receive the Department mailing, call Dr. Cundiff at 609-292-7300.

AMA DELEGATION



William J. D'Elia, M.D.

Last month brought a change to the AMA Delegation of the Medical Society of New Jersey. William J. D'Elia, M.D., stepped down as Chairman, yet is remaining on the Delegation. Karl T. Franzoni, M.D., has taken over the Chairman's responsibilities, and Frank Y. Watson, M.D., serves as Vice-Chairman.

TOBACCO LABEL WARNINGS

Dr. Frederick B. Cohen has called for warning labels on packages of chewing tobacco and tins of snuff. Citing mounting evidence that smokeless tobacco is just as harmful as smoking, Dr. Cohen says it makes a compelling case for warnings to be printed on all smokeless tobacco products, too.

Dr. Cohen, director of oncology at Newark Beth Israel Medical Center, notes "The culprits in smokeless tobacco are cancer-causing compounds called nitrosamines, which form in the mouth through the chemical interaction of saliva and tobacco." According to Dr. Cohen, "A plug of chewing tobacco or a pinch of snuff delivers roughly the same amount of nicotine as a cigarette and 10 times the nitrosamines."

Dr. Cohen urges support of two bills in Congress (H.R. 3510, and S. 1574) that would place warning labels on smokeless tobacco products. The labels would read:

- WARNING: This product may cause mouth cancer.
- WARNING: This product may cause gum disease and tooth loss.
- WARNING: This product contains nicotine and may be addictive.

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PROFESSIONAL LIABILITY COMMENTARY*

Liability Legislation and Tort Reform Savings

AMA Comments on Legislation; Potential Savings from Passage of Tort Reforms

AMA PROFESSIONAL LIABILITY LEGISLATION IN THE 99th CONGRESS

Three major bills are pending in Congress that seek to resolve the professional liability crisis: H.R. 2659, introduced by Representative Robert Mrazek (D-NY); H.R. 3084, introduced by Representatives Henson Moore (R-LA) and Richard Gephardt (D-MO); and S. 1804, sponsored by Senators Hatch (R-UT) and Inouye (D-HI). A fourth bill, S. 175, introduced by Sen. Inouye, also seeks to deal with professional liability problems and contains many concepts similar to those in the AMA-supported legislation. However, it has not received the same level of Congressional attention and discussion as the three other bills. The three bills and AMA position on them are summarized.

H.R. 2659 would provide federal financial incentives for states to establish malpractice panels with the exclusive original jurisdiction over medical malpractice claims with very limited appeal rights. Reforms such as eliminating the collateral source rule, frivolous claim penalties, limits on noneconomic damages, periodic payments, and attorney fee regulation are incorporated into the panel system in the bill.

The AMA supports the intent and objectives of H.R. 2659 to remedy the professional liability crisis. However, the AMA has certain concerns with potential constitutional questions raised by the screening panels encouraged by the bill.

H.R. 3084 would establish an alternative system for settlement of medical professional liability claims for injuries arising from the provision of health care services paid for or provided by Medicare, Medicaid, or any other program established under federal law. The central feature of H.R. 3084 permits a physician or hospital to offer compensation to any injured patient for that patient's "net economic loss" and by so doing foreclose the patient from suits for compensation in the court system. An arbitration provision in the bill permits a patient to initiate the tender offer process. Other provisions in the bill provide for automatic joinder of other parties by an offering party with contribution provisions to allocate payment of net economic loss between responsible parties.

The bill provides that its alternative federal program would not apply to otherwise covered injuries if a state has an alternative medical liability law in effect which is designated to bring about prompt payment for loss in the case of damages arising from the provision of health care services.

The AMA does not support H.R. 3084. It is not clear that the bill will reduce the upward pressures on malpractice insurance premiums, nor that it will reduce any incidences of the practice of "defensive medicine." Rather than reduce aggregate health care costs, the bill is likely to increase them and lead to higher total liability premiums and costs by encouraging more claims. Provision for joinder of parties in the bill would lead to unpredictable and possibly inequitable consequences.

S. 1804 would provide federal incentives for states to adopt professional liability reforms. Under the bill, a state would be eligible to receive federal grants if the following reforms are adopted by the state and made applicable to cases involving health care malpractice: (1) Mandatory periodic payments for awards of future damages exceeding $100,000. (2) Awards reduced by compensation received from other sources (eliminating collateral source rule). (3) Awards of noneconomic damages to be limited to $250,000. (4) Attorneys' fees to be limited to 40 percent of first $50,000 of award, 33.33 percent of next $50,000, 25 percent of next $100,000, and 10 percent of any award in excess of $200,000. In addition, to qualify for grants a state would have to adopt the following reforms: (1) Licensing fees from health care professionals would be allocated to the state agency responsible for disciplinary actions. (2) Hospitals would be required to have risk management systems. (3) Insurance companies in the state would be required to conduct risk management programs and require physician participation as a condition of maintaining insurance. (4) Insurance companies would be required to make data concerning malpractice awards available to state disciplinary agencies. (5) State law would have to provide for expanded peer review activities by state medical societies.

The AMA strongly supports prompt passage of S. 1804. The bill was developed by the AMA and its enactment is a number one priority of the Association. Passage of S. 1804 will provide added incentives to states to adopt needed reforms. States which have adopted these reforms have experienced an alleviation of critical professional liability problems.

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*This item from the Department of Professional Liability Control, MSNJ, was prepared by James E. George, M.D., J.D., and A. Ronald Rouse, who are, respectively, Director of the Department and Director of Special Projects.

POTENTIAL SAVINGS FROM PASSAGE OF TORT REFORMS

If four specific tort reforms are adopted universally by the states, the one-time savings in professional liability costs could total $800 million to $1 billion, says a recently released study by Milliman & Robertson of New York City for the American Medical Association.

The reforms—mandatory periodic payments for awards over $100,000; offset of awards and settlements by collateral source compensation; a $250,000 cap on noneconomic damages; and a descending scale of attorneys' contingency fees—all are key elements in an AMA-developed national professional liability reform bill.

The actuarial analysis concluded that, based upon total estimated 1984 medical professional liability premium costs of $2.7 billion, the potential savings from the four reforms could represent as much as 28 percent of the total malpractice costs in a state.

"Applied to the 1984 medical professional liability costs of $2.7 billion, the potential initial savings is approximately $800 million," said Allan Kaufman of Milliman & Robertson. "Applied to the estimated 1986 medical professional liability costs of $3.6 billion, the potential initial savings is approximately $1 billion."

Here are estimated savings which each specific tort reform could generate in a "typical" state:

• Periodic payments	6%
• Collateral source offset	8%
• Contingency fee limitation	9%
• Noneconomic damage limits	12%

Milliman & Robertson totes up the combined savings and gets a 28 percent potential savings. "The 28 percent is the average effect of multiplying the different savings from each reform," stated Kaufman. "You don't get this number by adding up the percentages of savings for each reform. It is the combined effect of all of them, taking some overlap into consideration."

The actuarial study stressed, however, that even if all four tort reforms were enacted in every state, other factors could affect the extent of premium reductions for physicians and hospitals.

"To realize the potential savings, it is necessary that the law impact claim settlements to the same extent as court-awarded claims, even though the statutory language only applies specifically to court awards. In the extreme case, if the law had no effect on settlements, the value of the savings when applied only to court awards would be approximately 5 percent."

Savings will vary from state to state depending upon the accuracy of certain assumptions made in the study. The potential initial savings might not be reflected fully in cost reductions immediately after passage of a state law, the firm said in further comment, because insurers and joint underwriting associations "might be reluctant to decrease rates by the full amount of potential savings until the effectiveness of the law could be tested."

The type of coverage offered also would affect the speed and extent of savings which could accrue. A claims made policy could reflect full initial cost savings within three to five years, but an occurrence polic might not show full initial cost savings for five to eigh years, the actuaries said.

The cap on noneconomic damages, which in th Hatch bill is set at $250,000, would be the legislativ action generating the largest savings. "We believe th reduction in trend over the 1986 to 1989 period fo a typical state will approximate 4 percent per year, wit most states realizing a trend savings ranging from to 6 percent. The trend reduction in the typical stat is equivalent to $80 million per year at 1984 cost level and $100 million at 1986 cost levels."

According to the New York firm, yearly savings woul continue to increase because professional liability settlement and awards costs are steadily rising and because of inflation.

Kaufman's firm hedged its bets by candidly statin that projecting potential savings rely on certain assumptions and judgments made in the study, but sai it considered the judgments "reasonable." Even so said the report, "other reasonable judgments could result in significantly different results."

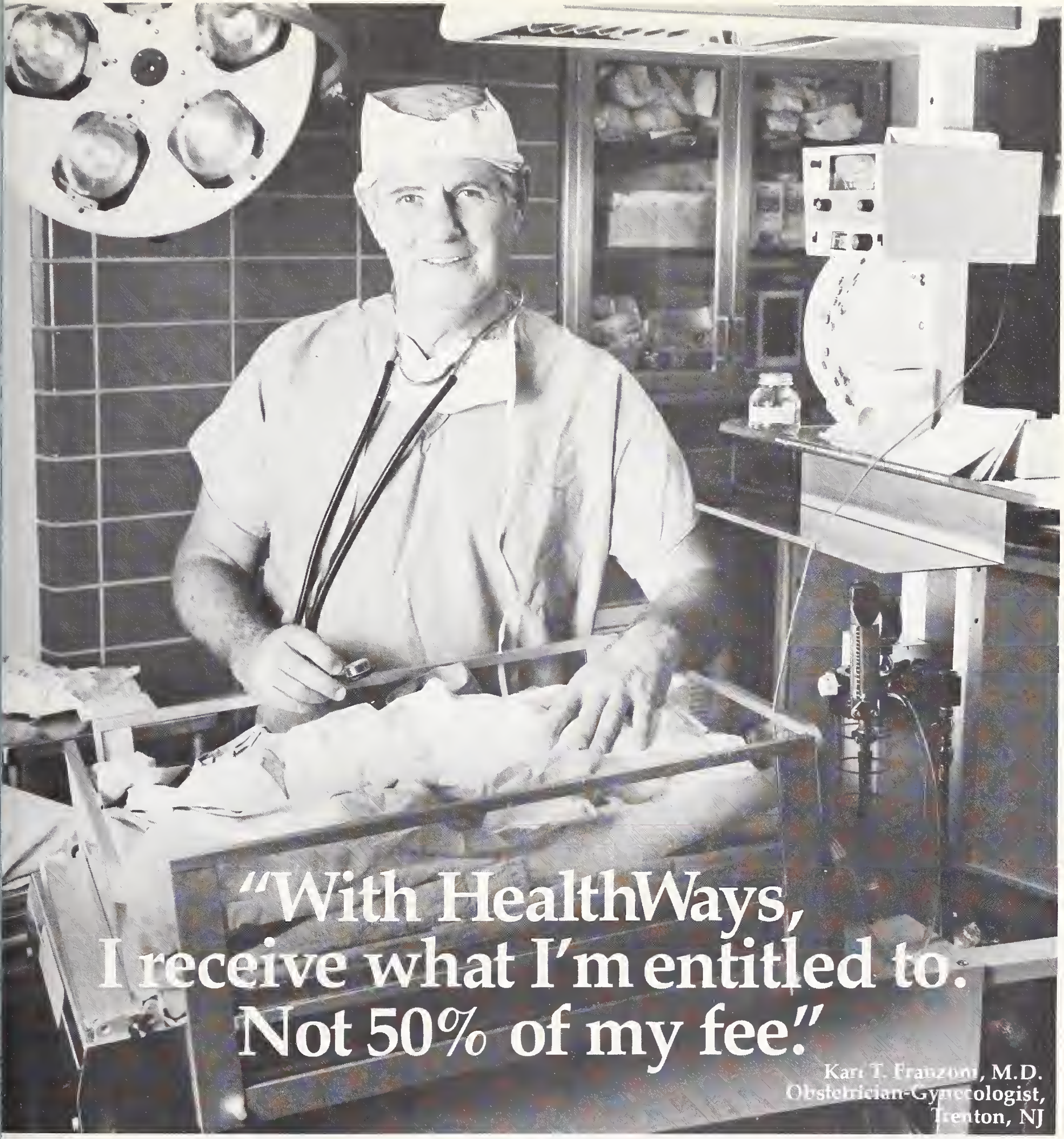
Further, actual savings will depend on "factors suc as plaintiff behavior, attorney behavior, and court interpretations which cannot be predicted in advance."

Similarly critical to savings outcomes is the application of any tort reform measures to settlements a well as to court awards. Right now, the statutor language in pending legislation applies only to cour actions.

To assure that no claimant is penalized by application of a collateral source offset, Milliman & Robertson suggested that professional liability insurer issue a health insurance policy to cover any gaps i benefits awarded by a court or agreed to in a settlement if collateral sources of those benefits becom unavailable in the future.

Data from *Best's*, joint underwriting associations patient compensation funds, and hospital self-insurance and captive insurance programs were used t calculate total professional liability premiums in th study. Award amounts for noneconomic damages wer assumed to equal 54 percent of the limited awar amount at 1974 to 1978 closed claim cost levels. ("safety factor" was calculated into savings related t the noneconomic damages and these savings reduce by 40 percent in view of unknowns.) Legal defens costs were assumed to equal 25 percent of indemnit amounts before the limitation on noneconomic damages. "We are assuming no savings in defense costs i this study," said Kaufman.

Milliman & Robertson also pointed out that if jurie correctly conclude future interest income (time valu of money), inflation, and mortality in making thei awards to successful plaintiffs, then passing a periodi payment law might not have any impact upon indemnity payments. (*Medical Liability Monitor*, Vol. 10, N 11, November 1985).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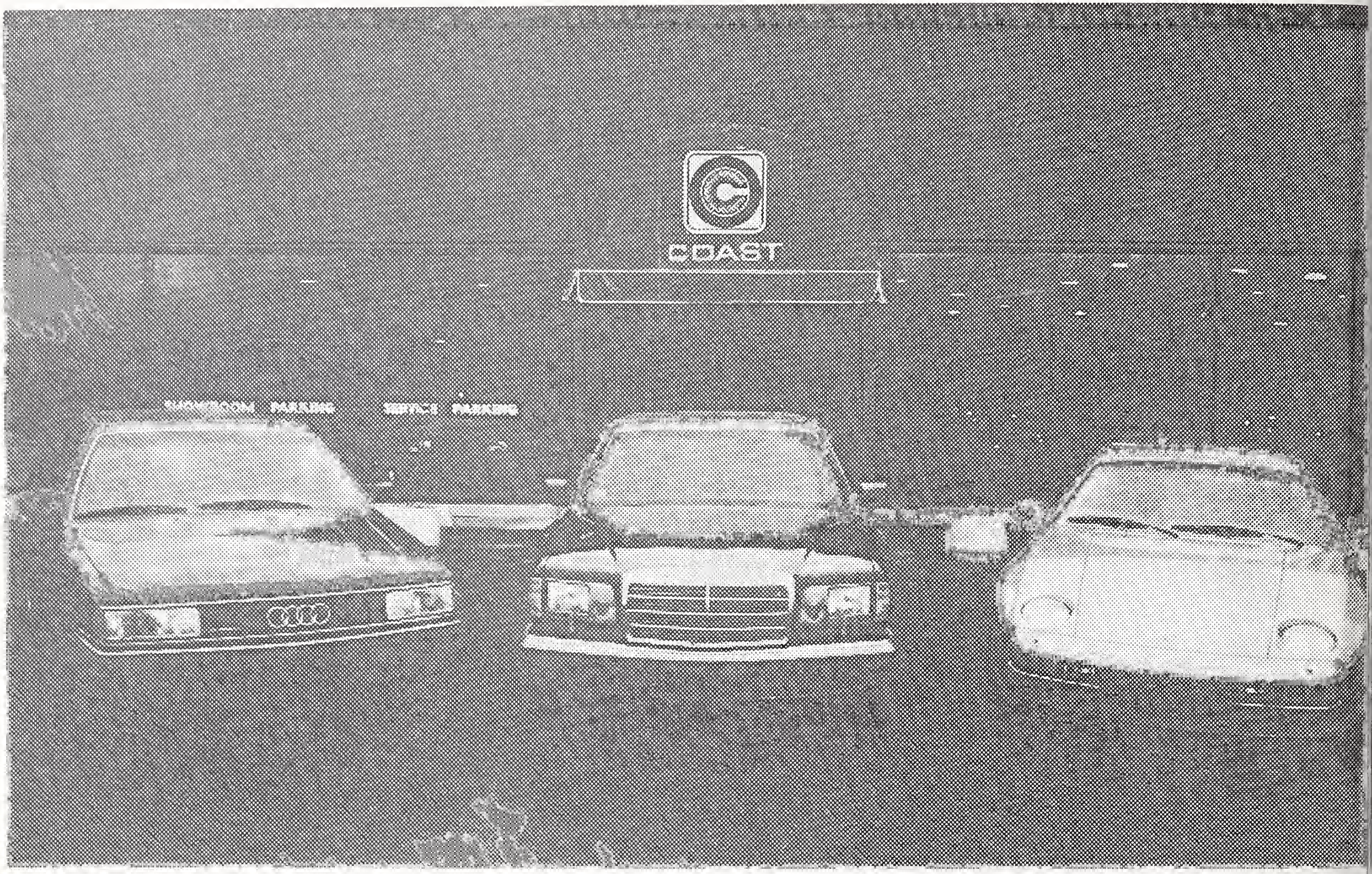

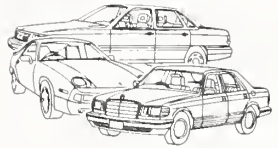

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PHYSICIAN AND PHARMACIST ATTITUDES TOWARD A GENERIC INCENTIVE PROGRAM

JOHN L. COLAIZZI, PH.D., AND JOSEPH A. BARONE, PHARM.D., PISCATAWAY*

New Jersey physicians and pharmacists were surveyed concerning a new dual copay prescription program designed to stimulate generic dispensing. They felt the program was restrictive and violated the spirit of the state generic substitution law.

During the 1970s, it became a policy of the federal government to encourage the use of generic drugs. Since 1980, the Food and Drug Administration has published a list of approved generically equivalent prescription products.[1] Generic substitution laws now have been implemented in all 50 states. With the increased prevalence of third-party prescription programs, there has been a greater desire on the part of program administrators to stimulate further generic drug usage in an attempt to minimize costs to the funding agency. A novel generic incentive program was introduced in New Jersey as a component of a Blue Cross prescription plan. In this program, patients are subject to a double copayment if available generic drug products are not dispensed. The copayment is a type of cost sharing whereby insured or covered persons pay a specified flat amount per unit of service, while the insurer pays the rest of the cost. The copayment is incurred at the time the service is used. Unlike coinsurance, which is payment of some percentage of the cost, the amount paid usually does not vary with the cost of the service.

STUDY METHODS AND RESULTS

From November to December 1984, physicians and pharmacists in New Jersey were surveyed in a single mailing to determine their views about the Blue Cross dual copay prescription program that had been implemented three to four months earlier. Questionnaires were mailed to 9,410 physician members of the Medical Society of New Jersey, which represents most physicians in the state and covers a wide range of specialty and general practice areas. A similar survey was mailed to the 3,450 pharmacist members of the New Jersey Pharmaceutical Association, including 68 percent independent retail pharmacists, 11 percent chain retail pharmacists, 8 percent hospital pharmacists, 6 percent industrially employed pharmacists, and 7 percent in other categories. The response rate to the questionnaire was 29 percent and 31 percent for physicians and pharmacists, respectively.

The responses indicated that the awareness level about the new program was much lower among physician respondents than among pharmacists (5 percent versus 70 percent), with most of the pharmacists having learned about it directly from Blue Cross or through the publications of their professional societies.

The respondents were asked how restrictive they felt this program (which carefully and succinctly was described in the survey instrument) would be on their professional prerogatives in selecting brand-name or generic prescription products. The responses are shown in Table 1. The results show that over two-thirds of the physicians and nearly nine out of ten pharmacists surveyed felt that the program either was excessively or moderately restrictive.

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*Dr. Colaizzi is Dean and Professor of Pharmacy and Dr. Barone is Assistant Professor and Director of Clinical Programs, Rutgers-The State University. Correspondence may be addressed to Dr. Colaizzi, College of Pharmacy, Rutgers-The State University, Piscataway, NJ 08854.

Physicians and pharmacists reacted rather similarly to the statement: "This program violates the spirit of the New Jersey generic substitution law which specifically permits physicians to exercise their professional prerogative to insist on a specific brand-name product." Their reactions are in Table 2.

TABLE 1

Answers to Questionnaire on Restrictiveness

Opinion	Physicians (%)	Pharmacists (%)
Excessively restrictive	37	67
Moderately restrictive	31	20
Slight	19	7
No effect	11	5
No response	2	1

TABLE 2

Answers to Questionnaire on Violating Spirit of Substitution Law

Opinion	Physicians (%)	Pharmacists (%)
Strongly agree	60	61
Agree	25	22
No opinion	6	4
Disagree	6	8
Strongly disagree	2	2
No response	1	3

TABLE 3

Answers to Questionnaire Concerning Copayments

Percent of Patients who Would Pay Double Without Complaining	Physicians (%)	Pharmacists (%)
0 to 19	18	56
20 to 39	20	20
40 to 59	24	12
60 to 79	20	5
80 to 100	13	5
No response	5	2

Physicians and pharmacists were asked to speculate on the likelihood that patients would be willing to pay the double copayment without trying to avoid such an extra payment by urging the physician or pharmacist to arrange for the substitution of the generic product, even in instances where the physician might prefer the brand-name product. Pharmacist respondents tended to anticipate greater problems with patient acceptanc of the higher copayment, as shown in Table 3.

As can be seen from this data, over half (56 percen of the pharmacist respondents felt a minimum of pa tients (0 to 19 percent) would not complain abou having to pay a double copayment, while only 10 pe cent felt that the majority of patients (60 to 100 pe cent) would not complain. Only 18 percent of phy sicians felt that the smallest number of patients (0 t 19 percent) would not complain, and 33 percent fe that the majority of patients (60 to 100 percent) woul not complain. Both pharmacists and physician agreed, however, that the elderly would be the mos likely to find difficulty with accepting the higher copay ment, followed by patients on long-term medication

Physicians were asked if they felt this progran would influence them to alter the rate at which the would specifically prohibit substitution on thei prescriptions. In a related question, pharmacists wer asked if they would be influenced to encourage phy sicians to permit substitution and/or patients to ac cept substitutes in cases where they had not done s before. The responses as shown in Table 4 demon strated that the program would likely have an impac on the actions of both groups to some extent.

When asked whether this program could impac negatively upon their relationship with patients, phy sicians responded somewhat less pessimistically tha pharmacists, as shown in Table 5.

TABLE 4

Answers to Questionnaire Concerning Influence on Permitting Substitution

Influence	Physicians (%)	Pharmacists (%)
To a major extent	11	20
To a moderate extent	29	20
To a slight extent	31	27
No effect	27	22
No response	2	3

TABLE 5

Answers to Questionnaire Concerning Impact of Program on Relationship with Patients

Opinion	Physicians (%)	Pharmacists (%)
Major negative impact	26	60
Minor negative impact	45	27
No effect	27	9
No response	2	4

When asked whether they felt some action should b taken to discourage such programs, 77 percent of th physicians and 89 percent of the pharmacists re sponded in the affirmative.

DISCUSSION

The administrators of third-party prescription pro grams generally feel that there has been a lack of sufficient incentive to stimulate physicians and pharmacists to choose generic drugs in filling third-party rembursed prescriptions. Approximately 50 percent of all new prescriptions offer the possibility of generic dispensing.[2] Various approaches are being used in third-party prescription programs to increase the rate of generic usage in an attempt to decrease costs to the ultimate agency responsible for funding the program. One approach provides a direct incentive to the pharmacist to select a generic by offering some type of financial bonus in the form of a higher dispensing fee, or a share in a portion of the cost savings resulting from the use of the generic product, or some other bonus to the pharmacist. More recently, programs directed toward the patient have been implemented, whereby the patient copayment is increased beyond the usual amount whenever a generic product is not used to fill the prescription. Whenever the potential exists to fill the prescription with a generic product, the patient will be penalized with an increased copayment, regardless of whether the decision not to use a generic product was the physician's, the pharmacist's, or the patient's.

By providing proper information, physicians and pharmacists will help patients understand the intricacies involved in the drug selection process.

In August 1984, Blue Cross of New Jersey announced the availability of a program in which some of their prescription program cardholders would have a dual copay option.[3] If such patients receive prescriptions for drugs which are substitutable under the New Jersey substitution formulary,[4] the patient must pay a double copayment ($4.00 instead of $2.00) if a lower cost generic form is not given. If, for example, the physician prescribes the drug by the brand name and indicates that generic substitution is not permitted, the patient will be subjected to the double copayment.

Although we currently are witnessing a marked increase in such consumer-directed incentive programs to increase generic substitution among third-party prescription programs within both the public and private sectors, there are some concerns among physicians and pharmacists about the implications of such programs. Although the patient usually is not in a position to assess the medical consequences of substitution in a given case, the financial penalty might cause the patient to bring undue pressure on the pharmacist or physician to substitute, possibly in instances where it might not be medically appropriate to do so. The dual copayment program also may violate the spirit of the state generic substitution laws. When initially drafted and implemented, various safeguards were incorporated universally into the statutes to make these laws acceptable to physicians and others. In most cases, for example, the physicians had the right to negate substitution. The financial pressure of dual copayment options sufficiently could influence some patients, with resultant diminution of the safeguards in the generic substitution laws. Such pressures on the drug selection process could be expanded easily from generic substitution to therapeutic substitution (the substitution of a drug with a similar therapeutic effect, but a different molecular structure and different generic name from the drug prescribed), with potentially greater risks. In 1985, legislation cleared the Iowa state legislature that would have allowed pharmacists to select therapeutic alternatives to prescribed drugs, when generally authorized by the prescriber.[5] Although subsequently vetoed by the governor, such legislative action could represent the beginning of a trend toward state-sanctioned therapeutic substitution.

CONCLUSIONS

Although programs designed to enhance the use of generic drugs might have a valuable intent of reducing health care costs, it is alarming that so many physicians and pharmacists harbor substantial reservations about such efforts to increase the rate of generic substitution, even though substitution legislation now exists in all 50 states. Furthermore, the rate of substitution (defined as the percent of times pharmacists substitute when it is possible to do so) is increasing and is approaching 50 percent or more.[6,7] The results of this survey clearly reveal that there is substantial concern among the physicians and pharmacists surveyed over the implications of the generic incentive program that was described. In addition, a comparative lack of awareness about such programs among physicians could pose problems whenever patients seek direction about the substitution of their prescription medications with generic products.

The view has been expressed that, in the long run, the increased use of generic drugs may not result in overall savings to the health care system since a two-tier drug price system may evolve—one for generics and the other for new drugs being introduced into the market.[8] This view reasonably assumes that pharmaceutical manufacturers will have to spread the cost of research and development for new products over the period of time during which the product remains on patent, since it no longer will be possible for them to recapture any part of this cost once the product goes off patent and enters the much more competitive market of multisource, generically available products. Those who cannot afford to pay for more expensive, newer drugs then may be unable to obtain "state-of-the-art" medicines. The availability of incentive mechanisms to place pressure on physicians, pharmacists, and patients to utilize generic medications, could mean that many patients would not easily obtain the more expensive medications in restrictive programs.

The rapid expansion of incentive programs and other similar programs that reflect the restrictions associated with cost containment are of concern to many pharmacists and physicians. While the intent of these programs has validity in terms of reducing health care costs, the results presented here demonstrate that many pharmacists and physicians also have concerns about the consequences of such programs. These concerns relate to increasing restrictions on professional prerogatives in the selection of appropriate prescription drug products. In some cases, it is possible that such restrictions could impact adversely on the drug therapy of some patients. The mechanisms inherent in such incentive programs could be applied with equal restrictiveness to the concept of therapeutic substitution, and this could pose additional dangers for some patients. When economic pressures in the drug selection process excessively restrict professional judgment, sound pharmaceutical and medical decisions may be clouded by financial decisions. In addition, such programs further may complicate the dispensing process for pharmacists and may confuse or alter the proper implementation of the state generic substitution laws. Physicians and pharmacists must give attention and thought to the issues involved in these programs and must make their views known to those who shape policy in the third-party program arena, as well as to patients. By providing proper information to patients, physicians and pharmacists will help them to better understand the intricacies involved in the drug selection process, thereby enabling them to make appropriate decisions that will influence the cost of drug therapy.

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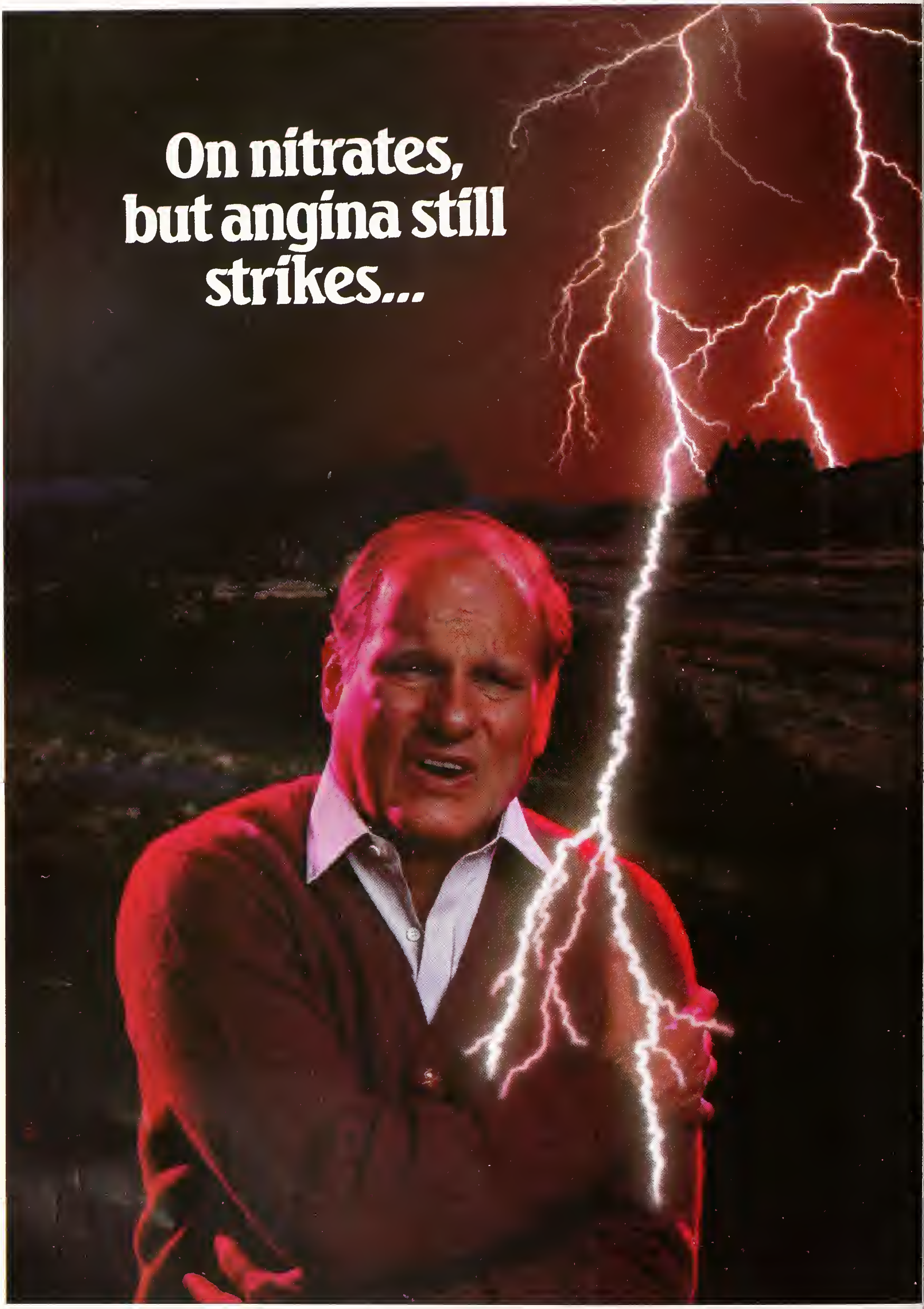
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Contraindications: Severe left ventricular dysfunction (see *Warnings),* hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (except in patients with a functioning artificial ventricular pacemaker), 2nd- or 3rd-degree AV block. **Warnings:** ISOPTIN should be avoided in patients with severe left ventricular dysfunction (e.g., ejection fraction $< 30\%$ or moderate to severe symptoms of cardiac failure) and in patients with any degree of ventricular dysfunction if they are receiving a beta blocker. (See *Precautions.)* Patients with milder ventricular dysfunction should, if possible, be controlled with optimum doses of digitalis and/or diuretics before ISOPTIN is used. (Note interactions with digoxin under *Precautions.*) ISOPTIN may occasionally produce hypotension (usually asymptomatic, orthostatic, mild and controlled by decrease in ISOPTIN dose). Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Such elevations may disappear even with continued treatment; however, four cases of hepatocellular injury by verapamil have been proven by rechallenge. Periodic monitoring of liver function is prudent during verapamil therapy. Patients with atrial flutter or fibrillation and an accessory AV pathway (e.g. W-P-W or L-G-L syndromes) may develop increased antegrade conduction across the aberrant pathway bypassing the AV node, producing a very rapid ventricular response after receiving ISOPTIN (or digitalis). Treatment is usually D.C.-cardioversion, which has been used safely and effectively after ISOPTIN. Because of verapamil's effect on AV conduction and the SA node, 1° AV block and transient bradycardia may occur. High grade block, however, has been infrequently observed. Marked 1° or progressive 2° or 3° AV block requires a dosage reduction or, rarely, discontinuation and institution of appropriate therapy depending upon the clinical situation. Patients with hypertrophic cardiomyopathy (IHSS) received verapamil in doses up to 720 mg/day. It must be appreciated that this group of patients had a serious disease with a high mortality rate and that most were refractory or intolerant to propranolol. A variety of serious adverse effects were seen in this group of patients including sinus bradycardia, 2° AV block, sinus arrest, pulmonary edema and/or severe hypotension. Most adverse effects responded well to dose reduction and only rarely was verapamil discontinued. **Precautions:** ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacologic effects. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, close surveillance of vital signs and clinical status should be carried out. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function. 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One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. *Pregnancy Category C*: There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use. **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia: HR < 50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%), elevations of liver enzymes have been reported. (See *Warnings.*) The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: ecchymosis, bruising, gynecomastia, psychotic symptoms, confusion, paresthesia, insomnia, somnolence, equilibrium disorder, blurred vision, syncope, muscle cramp, shakiness, claudication, hair loss, macules, spotty menstruation. **How Supplied:** ISOPTIN (verapamil HCl) is supplied in round, scored, film-coated tablets containing either 80 mg or 120 mg of verapamil hydrochloride and embossed with "ISOPTIN 80" or "ISOPTIN 120" on one side and with "KNOLL" on the reverse side. Revised August, 1984. 2385

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CENTRAL PONTINE MYELINOLYSIS: ROLE OF CT IN THE DIAGNOSIS

JAMES C. KOSS, M.D., EDWIN S. WILSON, M.D., NARNI GIRI, M.D., MOUNT HOLLY*

Central pontine myelinolysis (CPM) is a demyelinating disease manifested by confusion, cranial nerve dysfunction, quadriparesis, and hyperreflexia. High resolution CT scan is a good method for the early diagnosis of CPM.

Central pontine myelinolysis (CPM) was described by Adams, Victor, and Mancall in 1959.[1] Since then, more than 150 cases have been reported in the literature.[2] The typical clinical picture includes confusion, cranial nerve dysfunction, quadriparesis, and hyperreflexia.[3,4] Alcoholism and malnutrition have been implicated as risk factors. More recently, hyponatremia has been reported as a precipitating factor.[5]

Although the entity may have been suspected on clinical grounds prior to the advent of computed tomography, the diagnosis of CPM was seldom made prior to autopsy.[3] Several recent reports indicate that characteristic CT findings may occur in patients with CPM.[2,6,7] In the following case report, the striking computed tomography (CT) findings aided in the diagnosis of CPM.

CASE REPORT

A 27-year-old man with a 9-year history of severe alcoholism was admitted to the hospital with a five-day history of nausea, vomiting, profound lethargy, and generalized weakness. Physical examination revealed an afebrile, moderately dehydrated male. He was alert and responsive but had quadriparesis, hyperreflexia, and a right plantar extensor response. Bilateral facial muscle weakness was present. Laboratory studies revealed that the serum sodium was 138 mEq per liter, and the serum potassium was 4.4 mEq per liter.

A CT examination on the third hospital day demonstrated a well-defined area of decreased attenuation in the central area of the pons (Figures 1 and 2). The overall size of the pons was normal, with no suggestion of mass effect. The basilar cisterns were enlarged slightly. The cerebral cortical sulci were mildly prominent. There was no abnormal enhancement of the pontine lesion following the injection of contrast medium. On the basis of the characteristic CT findings together with the clinical presentation, the diagnosis of CPM was established.

On hospital day 10, the patient developed urinary incontinence and mild confusion. These findings resolved within several days. A followup CT examination was performed on hospital day 16, and there was no change from the initial study. Following 18 days of hospitalization, the patient was discharged with mild residual quadriparesis. The urinary incontinence and confusion had cleared.

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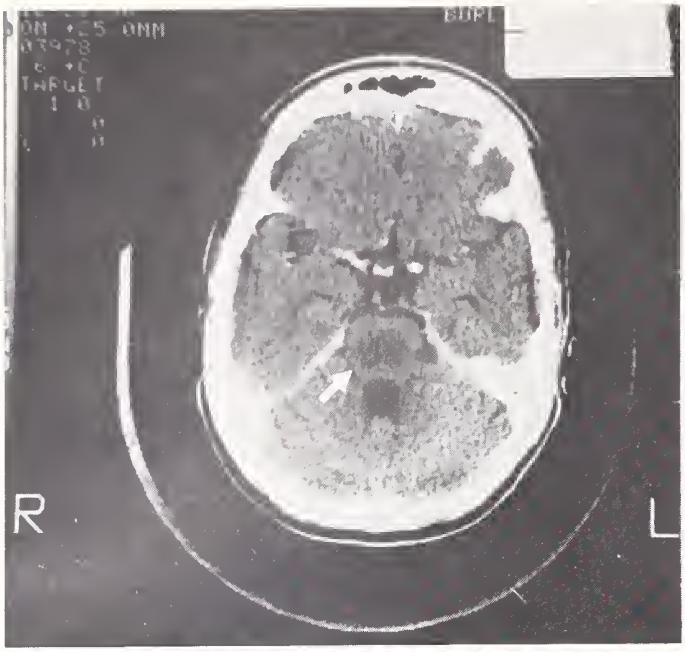

Figure 1—CT section through the level of the pons (arrow).

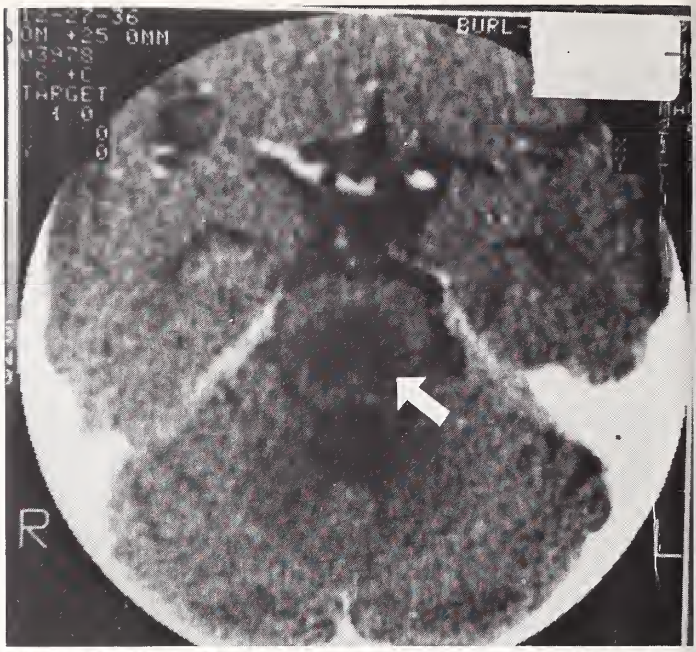

Figure 2—Note the hypodense area (arrow) in the central portion of the pons.

DISCUSSION

The finding of a low-density lesion without contrast enhancement within the pons during CT evokes the differential diagnosis of brain stem glioma, infarction, and demyelinating disease, including multiple sclerosis. The lack of CT contrast enhancement within the lesion mitigated against the diagnosis of multiple sclerosis. The young age of the patient made brain stem infarction extremely unlikely. Brain stem glioma may or may not exhibit contrast enhancement, but there generally is mass effect and pontine enlargement. These findings were not present in this patient. In fact, the brain stem size was normal to relatively diminished and the adjacent cisterns were slightly enlarged, which favored a degenerative and/or atrophic process. For all these reasons including the clinical presentation and history of alcoholism, CPM was the favored diagnosis.

Prior to CT, arteriography and pneumoencephalography were the primary imaging modalities for evaluation of suspected lesions of the brain stem. These procedures are not only invasive in nature but present major limitations in their ability to detect brain stem pathology. The central pontine lesion demonstrated in our case would not have been detected by any other imaging method prior to the development of high resolution CT.

CPM generally is considered to follow a usually fatal course.[5] This presumption reflects both the gravity of the disease and the prior difficulty in establishing the diagnosis prior to autopsy. CT has allowed the earlier and more precise clinical diagnosis of CPM. Of the seven previously reported cases of CPM confirmed by CT, four cases have survived.[2] This relatively high survival rate suggests that there may be a wider spectrum of clinical severity than previously was recognized.

The characteristic low-density lesion without enhancement on CT usually is limited to the ventral aspect of the pons, the basis pontis. When the process is very extensive, the tegmentum may become involved. There generally is a rim of isodense normal tissue at the periphery of the defect, even with the most extensive lesions.[8] Pathologic specimens at autopsy reveal that the myelin surrounding the axons has been destroyed.[8]

In a review of the literature, Messert et al.[5] found that 78 percent of the patients presented with a history of moderate to severe alcohol abuse. Of these patients, 88 percent had abnormal electrolytes and 57 percent had decreased serum sodium levels. Recent theory of the pathogenesis of CPM suggests that electrolyte imbalance, especially hyponatremia, may be a major predisposing factor.[5] Cerebral edema may result from electrolyte imbalance. This directly may damage myelin sheaths or produce a secondary effect due to compression of small blood vessels. The result of either process is myelinolysis. The anatomic "grid" arrangement of the corticobulbar, corticospinal, and pontocerebellar tracts among the pontine nuclear masses may make the pons especially vulnerable to this process.[5]

Messert et al.[5] have suggested that early diagnosis and treatment is essential if a fatal course is to be averted. Appropriate measures to control cerebral edema must be utilized. The goal of treatment should be directed toward reversal of the electrolyte disturbances and reduction of cerebral edema. Early diagnosis of CPM is critical. High resolution CT provides a good method for the early diagnosis of this disease process. The technique is noninvasive, readily available in most areas, and highly cost effective. Magnetic resonance imaging most likely will have a major role in the diagnosis in the future, but there have been no reported cases of CPM diagnosed by magnetic resonance imaging to our knowledge. A high index of suspicion should exist in patients with the symptom complex described, and early high-resolution computed tomography should be performed. As better understanding of the disease develops, an improved strategy for treatment may evolve to improve the outcome of this often fatal process.

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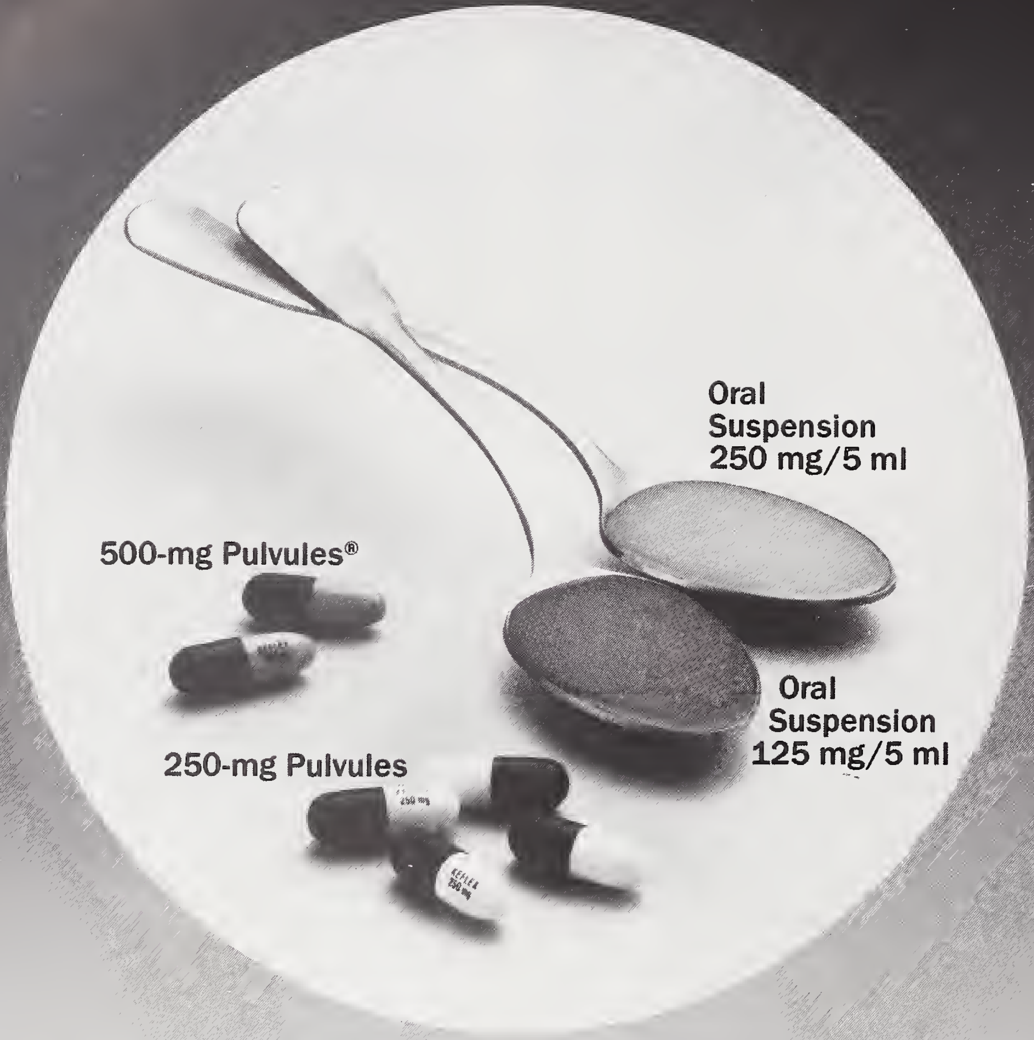

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REPORT ON THE NEW JERSEY POISON INFORMATION AND EDUCATION SYSTEM

STEVEN M. MARCUS, M.D., BRENT WHITEHEAD, PHARM.D., LOIS HONCHARUK, M.S.N., R.N., LEAH ZISKIN, M.D., ROY NICKELS, M.P.H., SALLY MATTHEWS, M.S., EDMOND DUFFY, JR., M.P.H., NEWARK*

The New Jersey Poison Information and Education System (NJPIES) has been operational for over two years. NJPIES handled over 42,000 telephone calls in 1984. This paper reports the epidemiological and experiential data for the first two years of NJPIES' operation.

A 22-year-old woman, despondent over social problems, called the New Jersey Poison Control Center (NJPCC) indicating she was about to commit suicide. After a lengthy conversation and her refusal to give her address or to go to a hospital for help, the Poison Center staff member patched in a "talk-down service." The talk-down service and the Poison Center staff were able to convince the woman to give her address. The staff member called the local police who responded to the emergency and took the woman to a hospital where appropriate therapy was given.

A 22-month-old child was bitten on the finger by a "pet" rattlesnake. The parents called the rescue squad for transport to a local hospital. After arriving at the home the rescue squad called the hospital which in turn called the Poison Center for advice as to proper handling of the case. The Poison Center determined that a sufficient quantity of antivenin was available at the Bronx Zoo. The decision was made to bypass the local emergency room and to transfer the patient to the Bronx Municipal Hospital Center. The Bronx Municipal Hospital staff is experienced in treating snake bites and is a few blocks away from the Bronx Zoo, thus, antivenin was easy to obtain. The child was treated without incident.

A 16-year-old child arrived at a local hospital after an apparent suicide attempt using an asthma medication which contained theophylline. The child was agitated and had a rapid heart rate but showed no additional symptoms. A theophylline blood level of 75 mcg/ml was found. The Poison Center was contacted. They advised the treating staff to use conservative measures. A nephrologist at the treating center also was consulted and suggested hemoperfusion for treatment of theophylline overdose. The Poison Center again was contacted when a repeat blood level did not show a significant drop over the course of two hours. Center staff suggested using multiple doses of oral-activated charcoal and cathartics rather than hemoperfusion. Poison Center treatment recommendations were followed and the blood level promptly dropped. The child did well without undergoing costly and dangerous hemoperfusion.

Each of these cases demonstrates ways in which the New Jersey Poison Information and Education System (NJPIES) has streamlined and improved the treatment of the acutely poisoned patient in the state of New Jersey. NJPIES was established in 1983 as the result

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Dr. Marcus is Project Director and Ms. Honcharuk is a Nurse Educator, New Jersey Poison Information and Education System. Dr. Whitehead is Director, New Jersey Poison Control Center. Dr. Ziskin is Assistant Commissioner, Mr. Nickels is Director, Office of Emergency Medical Services, Ms. Matthews is Coordinator, Health Project III (EMS), and Mr. Duffy is Coordinator, Accident Prevention and Poison Control, New Jersey State Department of Health. Correspondence may be addressed to Dr. Marcus, New Jersey Poison Information and Education System, 201 Lyons Avenue, Newark, NJ 07112.

TABLE
NJPIES Advisory Council

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- Rutgers Medical School: Department of Environmental and Community Health, Department of Pediatrics
- New Jersey School of Osteopathic Medicine
- Rutgers College of Pharmacy
- The Medical Society of New Jersey
- The Academy of Medicine of New Jersey
- New Jersey Chapter of the American Academy of Pediatrics
- New Jersey Hospital Association
- New Jersey Pharmaceutical Association
- New Jersey Coordinating Council
- Emergency Nurses Association

Month	1983 Calls average/day	1984 Calls average/day
January	0	106
February	31	106
March	51	107
April	72	109
May	95	116
June	84	126
July	83	118
August	90	113
September	99	120
October	103	122
November	109	115
December	107	120
Total	28,186	42,036

Figure 1—Call volume: average daily calls to New Jersey Poison Control Center.

of the New Jersey State Department of Health's Emergency Medical Services Poison Control Task Force report of August 1979. In this report, a task force of health care professionals and laymen reviewed the problems with the New Jersey's existent poison control program. In 1979, 34 centers located in acute care hospitals served as sources of information to the state. The centers covered only 17 of the 21 counties. With no clearly defined cachement areas and no formal designation, 6 centers existed in one county and 4 counties had no center. The true magnitude of the statewide problem could not be appreciated because the reporting from these centers was spotty, inconsistent, and difficult to analyze. In 1975, only 4,922 case reports were received by the New Jersey Department of Health; by population, at least 35,000 were expected. The task force concluded its report by suggesting that the state establish a regional poison information system for the entire state. The task force evolved into a working committee which formed an advisory council made up of members of the state's medical, medical education, nursing, pharmacy, and public health communities (Table).

This advisory committee founded NJPIES and submitted a grant request to the Consumer Products Division of McNeil Laboratories. NJPIES was awarded one of three grants in 1982.

In the fall of 1982, the New Jersey legislature adopted a bill calling for the establishment of a regional poison information system. In February 1983, five poison specialists, one Pharm.D. toxicologist, and one medical toxicologist began answering questions about acute and chronic intoxications from around the state of New Jersey. The hotline service of NJPIES became known as the New Jersey Poison Control Center (NJPCC) and receives calls at 1-800-962-1253.

This type of service has been provided over 70,000 times since February 2, 1983. In March 1985, the call

County	% Calls 1984	% Calls 1983	% Pop	% of Pop < 5 yrs.
Atlantic	3.6	3.4	2.6	2.5
Bergen	10.4	10.4	11.5	9.1
Burlington	2.8	1.8	4.9	5.3
Camden	9.3	6.4	6.4	7.4
Cape May	1.6	1.6	1.1	1.0
Cumberland	1.2	1.4	1.8	2.1
Essex	10.9	13.2	11.6	12.4
Gloucester	3.6	2.4	2.7	3.3
Hudson	5.0	5.0	7.6	8.1
Hunterdon	1.3	0.8	1.2	1.2
Mercer	3.6	4.4	4.2	3.9
Middlesex	6.2	6.2	8.1	7.5
Monmouth	9.6	9.4	6.9	6.8
Morris	5.2	5.3	5.5	5.2
Ocean	6.5	7.9	4.7	5.0
Passaic	5.8	6.4	6.1	6.5
Salem	0.9	0.8	0.9	1.0
Somerset	1.8	1.7	2.8	2.4
Sussex	2.3	2.8	1.6	2.1
Union	7.4	7.4	6.8	6.1
Warren	1.0	1.1	1.1	1.2

Figure 2—Calls received by New Jersey Poison Control Center by geographical area.

Age	1984	Subtotal 1984	1983	Subtotal 1983
<1 year	9.5%		10.3%	
1-2 years	23.1		25.5	
2-3 years	19.6		19.4	
3 years or less		52.2%		55.2%
3-5 years	10.2		9.3	
5 years or less		62.4		64.5
5-10 years	4.2		4.1	
10-20 years	8.6		7.7	
20-35 years	14.4			
35-55 years	7.1	20-60 years 21.8		
55-65 years	1.5	over 60 years 1.9		
>65 years	1.6			

Figure 3—1984 calls to New Jersey Poison Control Center by age of victim.

volume exceeded 140 calls each day (Figure 1).

Approximately 90 percent of calls received by NJPIES were related to toxic exposures while 10 percent were informational in nature. Eighty-five percent came from consumers and the remaining 15 percent came from health professionals, primarily in emergency rooms. Accessible by toll-free telephone services from anywhere within the state of New Jersey, the Poison Center now receives calls from all counties throughout the state. The calls to the Poison Center distribute according to population, not geography (Figure 2). In fact, many callers believe they are calling a local telephone exchange.

No seasonal, diurnal, or other pattern of call volume appears evident at this time. This may be due to the fact that the response to NJPIES still has not peaked. Whereas other poison centers appear to reach a plateau level and then have seasonal variations, NJPIES continues to grow.

As had been predicted by the Poison Center Task force, the majority of the calls to the Poison Center deal with children under the age of five. (Figure 3).

Calls relating to childhood exposures generally represent low level toxicities. In over 85 percent of the circumstances, these calls were handled safely at home, without the need for trips to hospital emergency rooms. An average New Jersey emergency room fee with no specific treatment rendered is in the neighborhood of $50. Thus, by calculating the number of visits to emergency rooms prevented by NJPIES, NJPIES has produced, in its first two years of operation, a savings of over two million dollars. All calls to NJPCC receive followup in the form of calls to ascertain the result of treatment. The hospitalized patients receive the most intensive followup calls from the Center.

Ten percent of the exposure-related, noninformational calls were handled with forced emesis with ipecac, activated charcoal, and cathartics. At no time were any complications noted. In over 95 percent of the episodes so managed, no further therapy was needed.

Less than 1 percent of the episodes reported to NJPIES developed life-threatening complications (Figure 4). There were 32 deaths reported during 1983 and 1984: 25 in humans, 7 in animals. All of the human deaths were the result of suicide with the victims dead or near-death on arrival at the treating center emergency room (Figures 5 and 6). Suicides represented 6.0 percent of the total calls and 7.6 percent of the calls

Effect	1984	1983
None	67.1%	80.0%
Minor	20.4	8.1
Major no sequelae	0.6	0.4
Major with sequelae	0.02(9)	0.03(5)
Expired	0.08(23)	0.07(12)
Lost to followup	11.7	11.4

Figure 4—Clinical outcome.

Number of Cases	Intoxicant
Human	
1	Acetaminophen
1	Adverse reaction to dental analgesia (chloral hydrate+Vistaril®+nitrous oxide)
2	Alcohol
1	Cocaine
1	Colchicine
1	Ethylene chloride
2	Ethylene glycol
1	Organophosphate insecticide
1	Percodan®
1	Demerol®
3	Tricyclic antidepressants
1	Unknown
Animal	
1	Carbamate insecticide
1	Piperazine
1	Sodium bisulfate pool pH adjuster
1	Soil acidifier/fertilizer
2	Unknown

Figure 5—Causes of death in 1984.

Number of Cases	Intoxicant
Human	
2	Antifreeze
1	Codeine and alcohol
1	Glutethimide and acetaminophen
1	Glutethimide and codeine
1	Oil of wintergreen
1	Amphetamines and gasoline
1	Cyanide
1	Amoxapine
Animal	
1	Copper

Figure 6—Causes of death in 1983.

when purely informational calls were excluded in 1984. This was down slightly from 8.0 percent in 1983.

More than 30 percent of the exposure-related calls concerned exposure to medications (Figure 7). Chief among these were aspirin, acetaminophen, and multiple vitamins. Over 20 percent of the calls were in response to exposures to household products, usually cleaning agents. Eight percent of the calls related to cosmetics. Generally, the cosmetic exposures were in children who ingested perfume, cologne, toilet water, or after-shave lotion.

	1984		1983
Intoxicant	**All Calls**	**No Info**	
Alcohols	2.2%	2.2%	3.0%
Bites/stings	1.4	1.4	0.5
Industrial products	1.7	1.5	*
Corrosives	1.4	1.4	1.3
Food	4.3	3.9	3.3
Foreign bodies	0.9	0.9	0.6
Gases/fumes	1.9	1.8	0.3
Home/office products	24.5	28.4	22.2
Solvents	3.4	3.5	0.5
Medicines	33.3	32.8	32.8
Metals	0.4	0.3	0.2
Pesticides	6.1	4.8	4.6
Plants	9.4	8.7	9.2
Others	9.1	7.4	21.5

*not stratified in 1983

Figure 7—Type of intoxicant involved in calls to New Jersey Poison Control Center.

The Poison Center received a large number of calls concerning pesticide exposure. Most of these related to three events: the ethylene dibromide (EDB) contamination of food, the use of chlordane as a termiticide, and the accidental venting of malathion by American Cyanamid. There were three severe poisonings from pesticides, all related to suicidal exposures.

NJPIES functioned as a back-up for the Department of Health handling inquiries from the public regarding such diverse problems as the nationwide recall of Zomax®, the crisis which developed from the discovery of dioxin contamination of soil surrounding Newark's Diamond Shamrock factory, EDB contamination of food, a malathion leak from American Cyanamid's Linden plant, and the problems with carbon monoxide contamination of private homes. In each instance the Poison Center's telephone number was published for the use of the public in obtaining information. In each case, the role of NJPCC was to field questions from the public and the media and thereby relieve various state agencies of this responsibility allowing them to operate more efficiently.

The volume of incoming calls to NJPIES continues to grow. The staff already has been expanded from an initial five poison specialists to ten. Each poison specialist is trained first in either medicine, pharmacy, or nursing and then receives special training before working at the hotline. The staff is backed up by a fellow in toxicology/pharmacology, a Pharm.D. in toxicology, and a full time-medical toxicologist. The Poison Center is staffed 24 hours a day, seven days a week, and possesses an extensive file of references related to a wide variety of substances.

NJPIES is funded by a contract from the New Jersey State Department of Health as a line item in the Department's annual budget. The amount of funds requested from the legislature is calculated to be sufficient to cover 80 percent of its costs. This represents the costs of the public health calls. Twenty percent of the yearly budget is derived from subscriptions paid by hospitals. The total cost of the program is estimated by extrapolating on the previous year's experience and adding a percentage for inflation and growth. Thirty-six of the acute care hospitals in the state became member hospitals in 1984. This was sufficient in 1984 to cover the complete cost of the program. In 1985, 44 hospitals have become members. Anticipated growth and demands on the program will provoke the need for additional funding from both the hospitals and the Department of Health.

Industry has not been responsive to the needs of the program. NJPIES sent requests for funds to over 5,000 corporations in New Jersey. The result barely covered the costs of the postage. With the exception of the original grant from McNeil Laboratories and small donations from several pharmaceutical companies to cover the cost of speakers for NJPIES meetings, only one pharmaceutical company, Hoffmann-La Roche, consistently has supported NJPIES but even its support has diminished over the past two years. Two chemical companies, Exxon Corporation and Exxon Research and Engineering have supported NJPIES with significant financial aid. The health insurance companies of the state, who have benefited most from the program in the terms of dollars saved, have responded poorly. Officials from The Prudential Insurance Company agreed to do all the printing needed by NJPIES but never produced a single piece. Blue Cross agreed that it should help support the program but the nature of this support has not yet been made known.

CONCLUSION

New Jersey has a regional poison information system of which it can be proud. It has earned a nationwide reputation with which few can compare. It can continue if funds can be found to continue its support. With such support, NJPIES will continue to provide exemplary help in the care of the poisoned patient.

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References:

1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

Reduces angina attack frequency*
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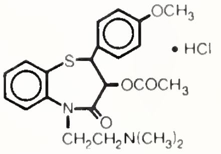
PROFESSIONAL USE INFORMATION

cardizem®
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy)-5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+)-cis-. The chemical structure is:



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect; cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given; a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS).

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1 asthenia (1.2%), AV block (1.1%). In addition, the following e were reported infrequently (less than 1%) with the order of pres tion corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, brad dia, palpitations, congestive heart fa syncope.
Nervous System:	Paresthesia, nervousness, somnole tremor, insomnia, hallucinations, and amn
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vom mild elevations of alkaline phosphatase, S SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensi
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episod vasospastic angina developed periods of transient asympto asystole approximately five hours after receiving a single 6 dose of CARDIZEM.

The following postmarketing events have been reported quently in patients receiving CARDIZEM: erythema multiforme kopenia; and extreme elevations of alkaline phosphatase, S SGPT, LDH, and CPK. However, a definitive cause and effect bet these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been lim Single oral doses of 300 mg of CARDIZEM have been well tole by healthy volunteers. In the event of overdosage or exagge response, appropriate supportive measures should be employ addition to gastric lavage. The following measures may be consid

Bradycardia	Administer atropine (0.60 to 1.0 mg). If is no response to vagal blockade, admir isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed degree AV block should be treated with diac pacing.
Cardiac Failure	Administer inotropic agents (isoproter dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarte bitartrate).

Actual treatment and dosage should depend on the severity c clinical situation and the judgment and experience of the tre physician.

The oral/LD_{50}'s in mice and rats range from 415 to 740 m and from 560 to 810 mg/kg, respectively. The intravenous LD_5 these species were 60 and 38 mg/kg, respectively. The oral L dogs is considered to be in excess of 50 mg/kg, while lethality seen in monkeys at 360 mg/kg. The toxic dose in man is not kr but blood levels in excess of 800 ng/ml have not been assoc with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic C nary Artery Disease or Angina Pectoris at Rest Due to C nary Artery Spasm. Dosage must be adjusted to each pati needs. Starting with 30 mg four times daily, before meals a bedtime, dosage should be increased gradually (given in di doses three or four times daily) at one- to two-day intervals optimum response is obtained. Although individual patients respond to any dosage level, the average optimum dosage appears to be 180 to 240 mg/day. There are no available data con ing dosage requirements in patients with impaired renal or he function. If the drug must be used in such patients, titration shou carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy**—CARDIZEM may be s coadministered with short- and long-acting nitrates, but have been no controlled studies to evaluate the antian effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 0088-1771-47) and in Unit Dose Identification Paks of 100 0088-1771-49). Each green tablet is engraved with MARION o side and 1771 engraved on the other. CARDIZEM 60-mg s tablets are supplied in bottles of 100 (NDC 0088-1772-47) and i Dose Identification Paks of 100 (NDC 0088-1772-49). Each y tablet is engraved with MARION on one side and 1772 on the

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PRIMARY MEDIASTINAL SEMINOMA PRESENTING AS SUPERIOR VENA CAVA OBSTRUCTION

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BENJAMIN SAFIRSTEIN, M.D., NEWARK*

Superior vena cava obstruction required multiple invasive biopsy procedures to establish its cause. Tissue was obtained showing noncaseating granuloma. We confirmed underlying malignant process by repeat open biopsy of the chest wall mass.

Carcinoma of the lung is the most common cause of superior vena cava obstruction. Other less frequent causes are lymphomas, aneurysms, and granulomatous disorder.[1-3] This paper documents the association of superior vena cava obstruction and primary mediastinal seminoma in a young homosexual male. Our purpose is to emphasize the rarity of this presentation and to describe the difficulties encountered in establishing a histologic diagnosis.

CASE REPORT

A 34-year-old white, homosexual male complained of facial swelling, headaches, and cough of three weeks' duration. He denied fever, weight loss, or anorexia. He does not smoke.

Physical examination revealed facial swelling and dilated jugular and anterior thoracic veins. No lymphadenopathy, hepatomegaly, or splenomegaly were found. The testes felt normal.

Hemoglobin was 14 gm/dl, white cell count was 5.600/mm^3 with a normal differential. A platelet count was 336,000/mm^3. Erythrocyte sedimentation rate was 93 mm/hr. Electrolytes, BUN, creatinine, blood glucose, and calcium were normal. Chest x-ray demonstrated an anterior mediastinal mass with hilar and paratracheal adenopathy.

Bronchoscopy was nondiagnostic. Scalene lymph node biopsy and mediastinoscopy demonstrated noncaseating granuloma. Despite this finding, we thought the patient had a malignancy, probably a lymphoma, because of his age, nonsmoking history, and homosexuality. This was supported by normal angiotensin-converting enzyme levels on three occasions. Therefore, we empirically treated the patient with radiotherapy in doses of 1,000 RADS to the mediastinum for palliation of symptoms. Complete radiographic and clinical clearing of lymphadenopathy occurred within two weeks. Kveim skin test was negative at four weeks. No further medications were given.

Sixteen months later, he complained of fever rising to 102° F., weight loss, anorexia, and dyspnea. Physical examination demonstrated a cachectic, dyspneic, male in acute distress. Examination revealed a mass measuring 5 x 10 cm over the left lateral-anterior chest wall. A massively enlarged tender liver was palpated. The testicles were normal. Chest x-ray demonstrated bilateral, multiple, large homogeneous masses and a large left-sided pleural effusion. Incisional biopsy of the chest wall mass and bone marrow biopsy revealed noncaseating granuloma. Pleural fluid was negative for acid-fast organisms and neoplastic cells. Repeat biopsy of the mass demonstrated sheets of malignant cells suggestive of malignant seminoma. A human chorionic gonadotrophin radioimmunoassay was elevated and consistent with tumor of trophoblastic origin. The patient was treated with cis-platinum, bleomycin, and vinblastine, responded well, and was discharged.

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Twenty months after his initial presentation, he was readmitted to the hospital and was found to have disseminated metastasis to bone and lung.

DISCUSSION

William Hunter in 1757 is credited for describing the first case of superior vena cava obstruction in a man with an aortic aneurysm.[4] In 1949, McIntire's extensive review of the literature showed that primary malignant thoracic tumors accounted for 33 percent of the cases and aneurysms and chronic mediastinitis for 30 percent of cases.[5] With the decline of tuberculous mediastinitis and syphilitic aneurysms, the majority of cases of superior vena cava obstruction were due to malignant thoracic tumors. The 1962 report of the Cleveland Clinic experience by Effler et al. stated that 80 percent of their cases were due to malignant neoplasms.[6] Banker et al. reported 438 cases of which 65 percent were due to primary lung cancer, 4 percent were due to Hodgkin's disease and lymphomas, and 7 percent were due to mediastinal malignancies.[7] Lochridge et al. reported 66 cases of superior vena cava obstruction of which 97 percent were due to malignancy, primary lung cancer being most common.[3] He reported only 2 cases of granulomatous mediastinitis, while lymphomas accounted for 12 percent of all malignant causes. Several authors have reported sarcoidosis presenting with vena cava obstruction.[8-11]

Our initial diagnosis was sarcoidosis after cervical and mediastinal lymph node biopsies demonstrated noncaseating granuloma. However, the asymmetric hilar adenopathy, normal angiotensin-converting enzyme level, negative Kveim test, and superior vena cava obstruction made sarcoidosis unlikely.

Following a brief remission of symptoms, our patient developed pleural effusion and a chest wall mass, which biopsy demonstrated to be malignant seminoma. Repeated testicular examinations were normal leading us to presume the seminoma arose in the mediastinum. Concomitant involvement of the anterior mediastinum and the testes is unusual, even in cases of disseminated testicular tumors.[12]

Extragonadal germ cell tumors are rare.[12] Seminoma is the most common cell type. Of 124 reports in the English literature, 14 reports presented with superior vena cava obstruction. Most of these tumors occur in the same age group as is affected by testicular primaries; like seminomas, they arise from a primordial germ cell. Primary mediastinal seminomas are extremely radiosensitive, therefore radiation therapy plays a major management role. The overall prognosis is worse for extragonadal seminomas than for primary seminomas, but it remains good with a 75 percent five-year survival rate. The mortality is highest within two years of diagnosis usually coincident with widespread metastasis.

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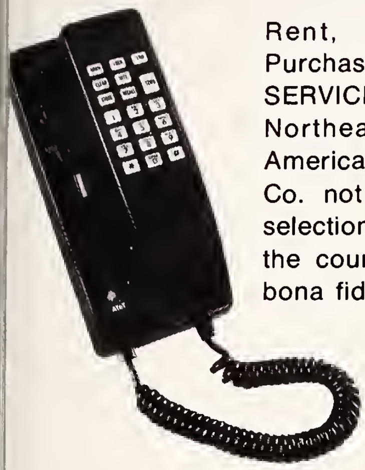

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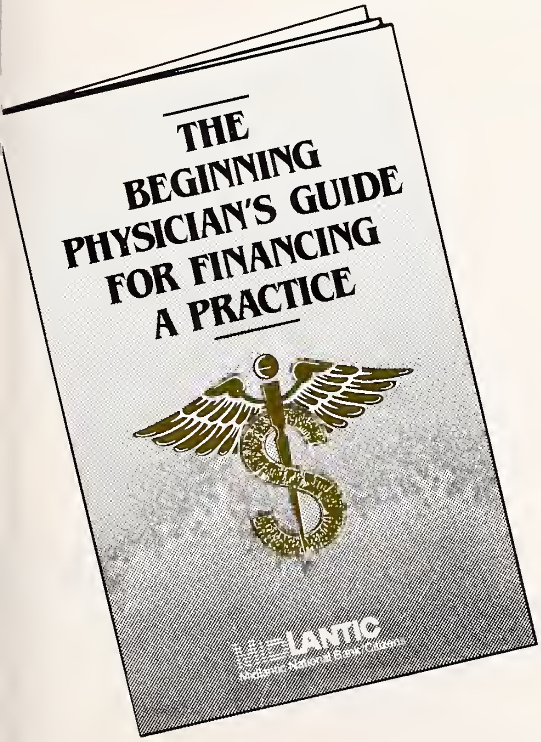

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SPECIALIZED NUTRITION SUPPORT IN NEW JERSEY UNDER THE PROSPECTIVE PAYMENT SYSTEM

MURRAY H. SELTZER, M.D., JOSEPH FLINTOSH, B.S., JAMES M. BLACKWOOD, M.D.,
C. CLAYTON GRIFFIN, M.D., NEWARK*

A survey was conducted to determine the effect of the DRG program on the existence of nutrition support teams and on the utilization of specialized intravenous nutrition support materials in New Jersey's acute care hospitals.

The national implementation of the prospective payment system via diagnostic related groups (DRG) in 1983 significantly has altered the approach by physicians, health care centers, and third-party payors to the provision of medical care and to the allocation of resources.

The DRG system, in rather vague terms, allows three categories for nutritional abnormalities. These very rarely are used by physicians to designate malnutrition as a primary disorder and only on occasion used to designate malnutrition as a comorbidity.

The Nutrition Support Committee of the Department of Surgery at the University of Medicine and Dentistry of New Jersey, Newark, has a particular interest in the effect of this new payment system on the utilization of specialized intravenous nutrition support materials and on the existence of nutrition support teams in the acute care hospitals of New Jersey.

MATERIALS AND METHODS

A questionnaire consisting of four statements requiring completion was mailed in 1985 to the acute care hospitals of New Jersey as listed in the Directory of the American Hospital Association.[1] The mailing was addressed to the director of the pharmacy in each institution anticipating that this person would be the most uniformly available person in each institution capable of and willing to complete the statements. The statements in the survey are listed in the Table.

RESULTS

Of the 96 hospitals polled, 67 (70 percent) replied. This response restated in terms of acute care hospital beds revealed that 21,345 beds out of 31,997 acute care beds (67 percent) were covered by this survey. Twenty-eight respondent hospitals had under 250 beds, another 28 hospitals had between 250 and 499 beds, and the remaining 11 hospitals had 500 or more beds.

Data Related to Statement 1. Fifteen (22 percent) of the respondent hospitals have nutrition support teams. In terms of acute care beds, 5,362 beds were covered by the teams, representing 17 percent of New Jersey's acute care beds (nonrespondents were assumed not to have a nutrition support team). Five nutrition support teams were present in hospitals having less than 250 beds, six teams were in hospitals having between 250 and 499 beds, and four teams were present in hospitals with 500 or more beds.

Data Related to Statement 2. Of those with teams, the DRG system was reported as being beneficial to 1 team, as detrimental to 3 teams, and as not having any effect on the remaining 11 teams. All 4 teams in the hospitals having more than 500 beds were unaffected by the DRG system. (The authors are aware of 1 hospital currently without a team attributing their loss of the team to the DRG system).

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Data Related to Statement 3. Sixty-three hospitals responded to the question regarding utilization of intravenous specialized nutrition support. Twenty-eight noted an increased utilization, 29 noted no change in utilization, and 6 noted a decrease.

When utilization of the intravenous nutrition support was correlated with the existence of a nutrition support team it was found that in those hospitals with a team, 6 noted an increased utilization, 6 were unchanged, and 3 were decreased. In those hospitals without a team, 22 noted an increased utilization, 23 were unchanged, 3 had a decrease in utilization, and 4 hospitals did not respond to statement 3.

DISCUSSION

A survey[2] of New Jersey's hospitals by one of the authors (MHS) in 1981 identified 10 functioning nutrition support teams. Thus, the currently identified 15 teams represent a 50 percent increase in teams over the past four years.

In evaluating our questionnaire, the assumption was made that none of the nonrespondent hospitals has a team. Although this certainly could be an erroneous assumption, most hospitals with a team are motivated to participate in such a survey. If indeed all teams responded, then 15.6 percent (15/96) of New Jersey's acute care hospitals have nutrition support teams. This number is to be compared with a very loose estimate[3] that 8.3% of all United States hospitals have teams. It has been reported by McShane and Fox[4] that 59.6 percent of nutrition support teams have been formed since 1980. It, therefore, becomes difficult to determine accurately the effect of the DRG system over a short time period. The increase in the number of teams, the increase in the utilization of intravenous specialized nutrition support in both hospitals with and without nutrition support teams, and the response by only 3 of 15 teams that the DRG system was detrimental leads one to conclude that the DRG system has not had a major influence on the formation of nutrition support teams and/or the utilization of specialized nutrition support in New Jersey. This conclusion must be a guarded one since it is too early to know if the rate of growth of new teams and the utilization of materials will be retarded in years to come by the prospective payment system. It is of concern to find increasing utilization of intravenous nutrition support without a commensurate growth in nutrition support teams to assure quality care associated with increased administration of such materials. Of the 67 hospitals reporting use of specialized intravenous nutrition support, only 15 have teams. It is assumed that many have oversight committees. It should be noted that the DRG system has imposed significant fiscal constraints on hospitals and one only can wonder if the rate of new teams being developed and the utilization of specialized nutrition materials would have been higher in the absence of the prospective payment system.

In conclusion, it appears that at this time New Jersey's acute care hospitals are providing specialized intravenous nutrition support at an increasing rate and that the provision of such care has not been affected adversely by the DRG system.

TABLE

Questionnaire

1. We have a Nutrition Support Team consisting of a physician, pharmacist, nurse, and dietitian.
   Yes ______ No ______
2. The DRG reimbursement system has helped impaired ______ not affected ______ the functioning of our team.
3. Utilization of Intravenous Nutrition Support Materials since the advent of DRG's is increased decreased ____ unchanged ____ at our institution.
4. Name of Hospital: ______
   Number of Beds: ______

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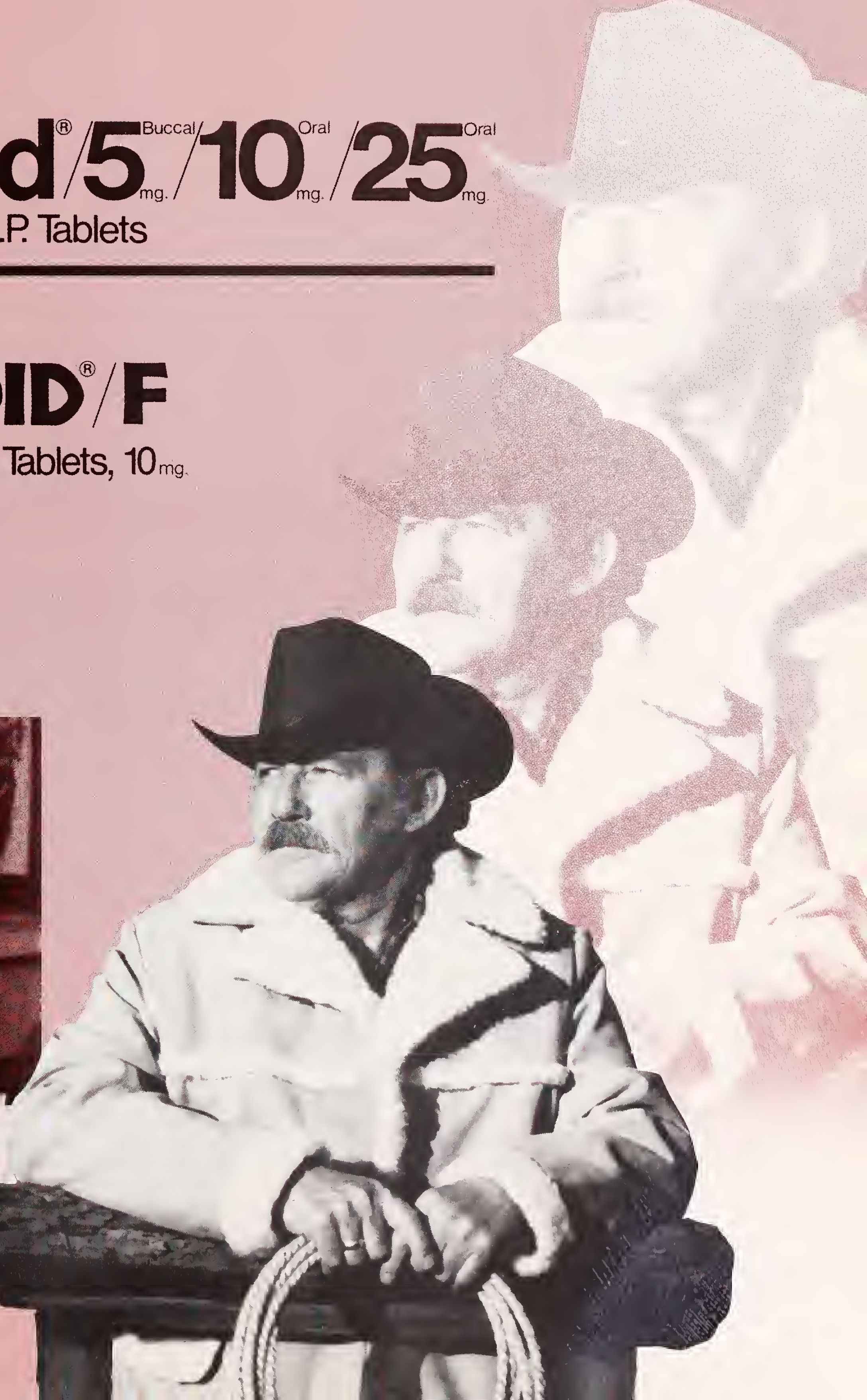

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CLINICAL NOTE: SMALL BOWEL INCARCERATION IN A DRAIN SITE HERNIA

JOHN WRABLE, JR., M.D., TOMS RIVER*

My patient developed an incarcerated hernia in a drain site 11 years after cholecystectomy. It was repaired successfully. Signs and symptoms were pain and a tender mass under the drain site scar.

Placement of drains in the subhepatic space after biliary tract surgery is common practice. On occasion, this procedure may be followed by complications from the surgery or after removal of the drain.[1,2,4] This report documents an example of the latter complication.

CASE REPORT

A 43-year-old female came to our emergency department complaining of right upper quadrant pain, nausea, and vomiting for 18 hours. Her abdomen was distended and a tender mass was felt in the right upper quadrant beneath a drain site scar. She had a cholecystectomy 11 years earlier through an upper midline incision. The penrose drain, which was used, was exited through a separate stab wound in the upper right quadrant.

I had previously seen this patient because of crampy pain and pain at the drain site. Because of her obesity, I was unable to feel a mass or abdominal defect.

At the operation, a loop of small bowel and omentum were found incarcerated in a 3 cm defect in the abdominal wall, 6 cm from the original incision. The intestine and omentum were freed and the abdominal wall hernia was repaired. Her postoperative course was uneventful.

DISCUSSION

The occurrence of abdominal drain site hernias with intestinal incarceration is rare. This complication at a remote site from the original surgical incision only has been reported on two other occasions.[3] Drains in abdominal surgery commonly are used for sepsis or if leakage is suspected. Placing a drain through the primary abdominal incision predisposes a patient to wound hernias, therefore customary practice is to exit the drain through a separate stab wound in the abdominal wall. Neither Hardy[1] nor Hermann[2] makes reference to this complication of abdominal drains. The first reported cases were in 1978 by Mosely who documented two cases of incarcerated drain site hernias remote from the primary incision and from the drain removal from the abdomen.[3] There have been reports of four cases of small bowel prolapse and/or incarceration in the immediate postremoval period requiring small bowel resection; two of these patients died.[3,4]

Pain, palpable mass, and tenderness under a drain site scar should alert the physician to a possible small bowel incarceration.

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*From the Department of Surgery, Community Memorial Hospital, Toms River. Correspondence may be addressed to Dr. Wrable, 20 Hospital Drive, Toms River, NJ 08753.

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DOCTORS' NOTEBOOK

UMDNJ Notes; AMNJ Report; Physicians Seeking Location in New Jersey

UMDNJ Notes

Stanley S. Bergen, Jr., M.D.
President

Seventy-six years ago, the Report on Medical Education in the United States generated the reformation of American medical education. The report, written by Abraham Flexner and published by the Carnegie Foundation for the Advancement of Teaching, had a fundamental influence on 20th century medical education.

But, the power is shifting and the roles are changing. Indeed, we are witnessing another reformation that is dictating how we will educate future physicians and how we will provide health care to patients. I speak of the transition from an era of medicine controlled by health care providers to one dominated by health care consumers. The reasons are simple: cost, access, and quality.

The federal government and the private sector desire lower costs, and, as a result, a new medical economics is taking hold, with private health care corporations as the anchor. Whether HMOs or investor-owned hospital corporations, these businesses are in intense competition for customers through price and convenience. Moreover, since there is reduced government support for medical education and biomedical research, medical training is moving from sophisticated teaching hospitals to lower overhead community hospitals, HMOs, and satellite clinics, while research is being funded by corporations.

In my annual State of the University Address, I outlined a number of objectives aimed at preparing UMDNJ for the new health care environment and, in so doing, establishing ourselves as one of the nation's top-ranked academic health sciences systems.

In 1986, we will seek new and strengthened partnerships with the state, private corporations, and hospitals to devise joint solutions to health care financing and delivery issues and other dilemmas posed by the changing marketplace.

But there is much of our present health care system—pared of its excesses—that must be protected. As New Jersey's health sciences university, we will preserve our University's precious traditions as a health professions education and research leader; an innovator of advanced and new specialized forms of care; and an advocate of the duty and responsibility that society has to meet the health care needs of the cities and the medically indigent. With an eye on cost-consciousness and efficiency, we are determined to see that the medical university remains the center of the medical profession.

Therefore, UMDNJ will dedicate its efforts to:

- Establish several endowed chairs to attract world-class individuals who will contribute to the margin of excellence the University is pursuing;
- Create a major cardiovascular research institute, the foundation of which will be the multifaceted cardiac research, diagnosis, and treatment programs underway at the Piscataway/New Brunswick campus of UMDNJ-Rutgers Medical School;
- Initiate university-wide research ventures in the areas of chronic pain, child development, and environmental medicine—toward the goal of bringing individual research projects into specially funded and coordinated programs;
- Bolster equipment and research facilities at the New Jersey Cancer Center in Newark, thereby providing better care for cancer patients and improved resources for physicians trained to give this specialized care;
- Increase mental health services, with special emphasis on teenagers;
- Provide more scholarships, fellowships and other forms of aid to UMDNJ students.

While these initiatives exemplify the quick and dramatic changes that academic medical institutions are making in research, patient care, and education, we must do more than merely adapt in the era of new medicine if we are to lead the way.

UMDNJ also needs greater autonomy and will work more closely with the state to develop the financial means for recruiting top educators, researchers, and administrators, and to gain primary consideration for UMDNJ and its core teaching hospitals in the state's certificate of need process to facilitate and make cost effective the acquisition and deployment of state-of-the-art equipment and the development of appropriate new specialized care services. We also intend to establish University Hospital as the state's major teaching and specialized care referral center by ensuring appropriate reimbursement rates for its specialized services, and by seeking other budgetary support, to make sure that teaching and indigency care costs no longer hinder the hospital's ability to mature.

AMNJ Report

Dennis R. Fillipone, M.D.
President

The Academy's popular Roving Symposia series has expanded out of the hospital format and into the television satellite arena. On January 30, 1986, Dr. Victor Parsonnet discussed "Current Trends and Issues in Cardiac Surgery" on the Healthcare Information Network (HIN) in Princeton. This satellite teleconference was followed on February 6, 1986, by Dr. Arthur Krosnick who discussed "Management of Diabetic Patients Undergoing General Surgery." HIN presently is serving 60 hospitals in New Jersey, and over 200 hospitals outside of the state participate in their programming. We hope to provide speakers on a monthly basis from the Academy's distinguished Speaker's Bureau.

On Saturday, March 15, 1986, the Academy will present two major symposia on topics of current interest. The Newark Airport Marriott will host a conference on "New Frontiers in Liver and Biliary Tract Radiology" which will be cosponsored by the Radiological Society of New Jersey. Among the distinguished faculty will be Dr. Fenton Schaffner from Mount Sinai School of Medicine and Dr. Kenneth Taylor of Yale University School of Medicine.

The New Jersey Medical School in Newark will be the site of the second meeting on "Management of Head Trauma in the 1980s." The purpose of this symposium is to provide an opportunity for health care providers to meet and discuss developments and controversies in the rapidly changing field of head trauma management. A large faculty has been assembled and includes Dr. Paul Cooper from SUNY-Downstate, Dr. Kenneth Shapiro from Albert Einstein in the Bronx, and Dr. Abbott Krieger, Chief of Neurological Surgery at UMDNJ-New Jersey Medical School.

The Academy of Medicine of New Jersey's Long-Range Planning Committee met on December 11, 1985, at the Loew's Glenpointe Hotel in Teaneck.

Physicians Seeking Location in New Jersey

The following physicians have written to the Executive Offices of MSNJ seeking information on possible opportunities for practice in New Jersey. The information listed below has been supplied by the physician. If you are interested in any further information concerning these physicians, we suggest you make inquiries directly to them.

EMERGENCY MEDICINE—Julian A. Sellek, M.D., 1501 South Greenway, Coral Gables, FL 33134. Salamanca (Spain) 1963. Also, family practice. Board eligible. Board certified (FP). Group, solo, partnership. Available.

FAMILY PRACTICE—Lawrence S. Gordon, M.D., 1016 Spinnaker Way, Apt. G-1, Melbourne, FL 32935. Rutgers 1982. Board certified. Group, partnership, solo. Available.

Natalia Moishezon, M.D., 207 Christie St., Leonia, NJ 07605. Moscow 1964. Board eligible. Group, partnership, industry. Available.

Jeffrey C. Schultz, M.D., 301 Victoria Dr., Monroeville, PA 15146. University of Massachusetts 1983. Board eligible. Group. Available July 1986.

Julian A. Sellek, M.D., 1501 South Greenway, Coral Gables, FL 33134. Salamanca (Spain) 1963. Also, emergency medicine. Board certified. Board eligible (EMER). Group, solo, partnership. Available.

Lew Herbert Valliant, M.D., 115 Mountain View Dr., Elkins, WV 26241. Queen's University (Canada) 1976. Board certified. Solo or group. Available.

GASTROENTEROLOGY—Richard J. Fastiggi, M.D., 10 Silver Stirrup Ct., Timonium, MD 21093. UMDNJ 1977. Board certified (IM). Solo, partnership, group. Available July 1986.

Prasad Satyanarayana Maturu, M.D., 8863 17th Ave., Brooklyn, NY 11214. Mysore Medical (India) 1977. Board eligible. Group or partnership. Available June 1986.

Brian C. Weiner, M.D., 24 Bowling Green Pl., Staten Island, NY 10314. SUNY-Downstate 1981. Board certified. Group or partnership. Available July 1986.

INTERNAL MEDICINE—Walter M. Bakun, M.D., 199 Casterline Rd., Den-

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May 1-4, 1986
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Wednesday, April 30, 1986
3:30 P.M. —Board of Trustees' meeting

Thursday, May 1, 1986
7:30 A.M. —Registration open
9:00 A.M. —Message Center and Exhibits open
2:00 P.M. —House of Delegates
4:00 P.M. —Reference Committees

Friday, May 2, 1986
8:00 A.M. —Registration, Message Center, and Exhibits open
9:00 A.M. —House of Delegates (election)
12:00 NOON —Golden Merit Award Ceremony followed by Reception
2:30 P.M. —Reference Committees
5:00 P.M. —JEMPAC Political Forum
5:45 P.M. —JEMPAC Wine & Cheese Reception

Saturday, May 3, 1986
8:00 A.M. —Registration, Message Center, and Exhibits open
8:30 A.M. —House of Delegates
1:30 P.M. —House of Delegates
6:30 P.M. —Inaugural Reception followed by Inaugural Dinner

Sunday, May 4, 1986
8:00 A.M. —Registration and Message Center open
8:30 A.M. —General Session—"Corporate Invasion in the Practice of Medicine"
1:00 P.M. —Board of Trustees' meeting

ville, NJ 07834. St. George's University (Grenada) 1983. Board eligible. Group, partnership, solo. Available July 1986.

Joseph Cohen, M.D., 16 Mountainbrow Village, Corning, NY 14830. New York Medical College 1982. Board eligible. Group. Available July 1986.

Arkady S. Fishman, M.D., 55 Nagle Ave., Apt. 1E, New York, NY 10040. Aktyubinsk Medical Institute 1969. Board certified. House staff, group, nursing home. Available.

Serge I. Kaftal, M.D., 125 Gates Ave., Apt. 38, Montclair, NJ 07042. Lodz Medical Academy (Poland) 1978. Solo, group, health center service. Available.

H. Homayouni, M.D., 459 Hamilton Pl., Hackensack, NJ 07601. Tehran (Iran) 1971. Board certified. Partnership. Available.

Rey Chi Lin, M.D., 714 Indian Creek Dr., Wilkes-Barre, PA 18702. China Medical 1977. Board eligible. Group, partnership, solo. Available July 1986.

Makkena Ramachandra Rao, M.D., 501 Indian Creek Dr., Wilkes-Barre, PA 18702. Guntur Medical (India) 1974. Board eligible. Group or partnership. Available July 1986.

Marian Lynn Taylor, M.D., 7 Loriann Rd., Warren, NJ 07060. UMDNJ 1983. Board eligible. Group or partnership. Available June 1986.

Dipak Vora, M.D., 509 Indian Creek Dr., Wilkes-Barre, PA 18702. University of Bombay (India). Board eligible. Available July 1986.

OPHTHALMOLOGY—Kenneth R. Brein, M.D., 55 Beacon St., Pittsburgh, PA 15217. University of Pennsylvania 1982. Board eligible. Group, partnership, solo. Available July 1986.

Andrew R. Gewirtz, M.D., 64 Pond Rd., Apt. G3, Woodbury, NY 11797. Chicago 1978. Board eligible. Board certified (PED) Single or multispecialty group. Available.

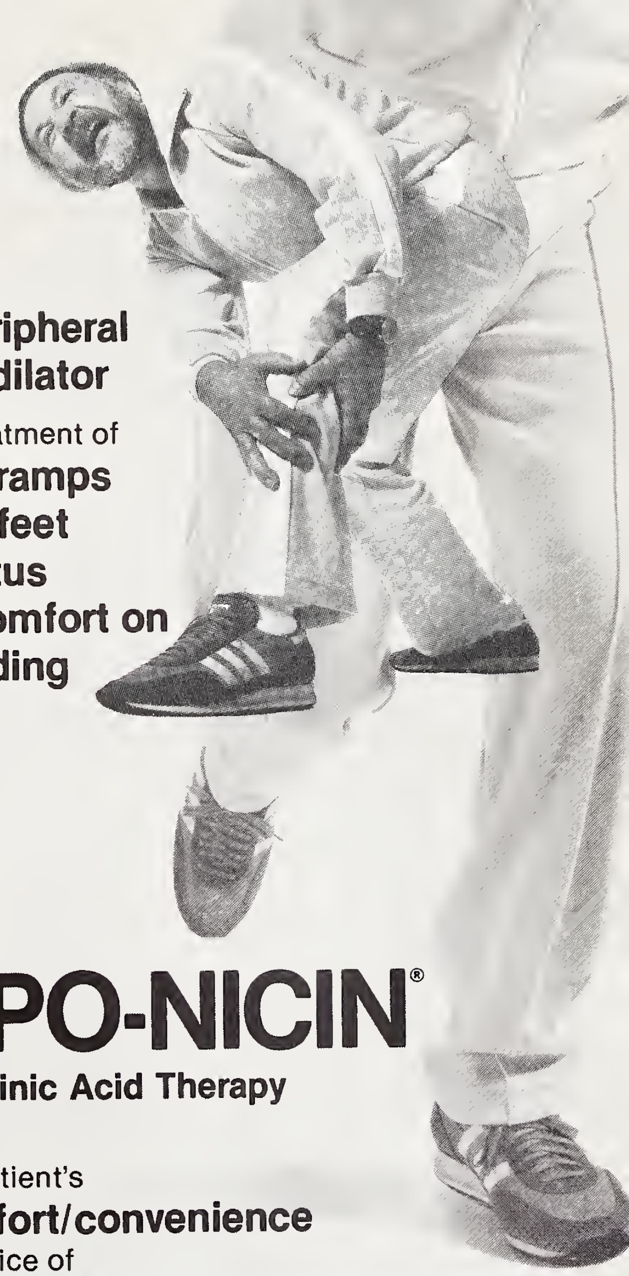
PULMONARY—Fred A. Wagshul, M.D., 55 Mill Plain Rd., Unit 16-4, Danbury, CT 06811. Universidad Central del Este 1980. Board eligible. (IM). Group, partnership, hospital-based. Available July 1986.

RADIOLOGY—Jesus S. Santos, Jr., M.D., RD 2, Gibson Rd., Goshen, NY 10924. Santo Tomas (Philippines) 1960. Board certified. Available.

SURGERY—Derrick John Martin, M.D., 13-2 Granada Cres., White Plains, NY 10603. West Indies 1977. Board certified. Group, partnership, solo. Available.

Russell H. Zelman, M.D., 309 DeMott St., Mineola, NY 11501. Universidad del Noreste (Mexico) 1980. Board eligible. Group, solo, partnership. Available July 1986.

UROLOGY—Louis C. Galdieri, M.D., 1563 Hampton Knoll Dr., Akron, OH 44313. Guadalajara (Mexico) 1979. Group or partnership. Available August 1986.



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Each time-release capsule contains:
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in a special base of prolonged therapeutic effect.
DOSE: 1 to 2 tablets daily.
AVAILABLE: Bottles of 100, 500.

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DOSE: 1 to 3 tablets daily.
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Americana Host Farm Resort

HOUSING APPLICATION
220th ANNUAL MEETING
THE MEDICAL SOCIETY OF NEW JERSEY

THURSDAY, MAY 1, TO SUNDAY, MAY 4, 1986

THE FOLLOWING ARE ALL-INCLUSIVE PACKAGE PLANS BASED ON MODIFIED AMERICAN PLAN, INCLUDING DINNER AND BREAKFAST DAILY STARTING WITH DINNER ON THE DAY OF ARRIVAL, THE SATURDAY NIGHT BANQUET, LIVE CABARET SHOWS EACH EVENING FEATURING A DIFFERENT SHOW EVERY NIGHT, STATE SALES TAX, AND ALL GRATUITIES COVERING BELLMEN FOR IN/OUT LUGGAGE SERVICE, CHAMBERMAIDS, AND ALL MEAL SERVICE AS OUTLINED:

FOUR-NIGHT PACKAGE
$435.00 PER PERSON, SINGLE OCCUPANCY
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- Forms received after April 1, will be confirmed on an "if avail basis.
- Fill out only one form for each required.
- DEPOSIT OF $100 PER R MUST ACCOMPANY FORM CONFIRM RESERVATIONS.
- Please make check payable to FARM. Major credit cards acc at hotel, but not for deposit.
- Hotel confirmation is require check-in.
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Host Reservations
2300 Lincoln Highway Eas
Lancaster, PA 17602
717-299-5500

CHECK TYPE OF ROOM REQUIRED: ☐ SINGLE OCCUPANCY ☐ DOUBLE OCCUPANCY

PLEASE NOTE: Number of persons attending the Inaugural Dinner-Dance honoring President-Elect and Edward A. Schauer on Saturday, May 3, 1986 ____________

PLEASE PRINT OR TYPE THE FOLLOWING INFORMATION:

NAME: ____________________ NO. of CHILDREN: ________

SHARING ROOM WITH: ____________________ CHILDREN'S AGES: ________

ADDRESS: ____________________

CITY: ____________________

PHONE: __________ STATE: __________ ZIP: __________

CHECK IF OFFICIAL DELEGATE ☐ COUNTY: __________

ARRIVAL DATE __________ DAY __________

DEPARTURE DATE __________ DAY __________

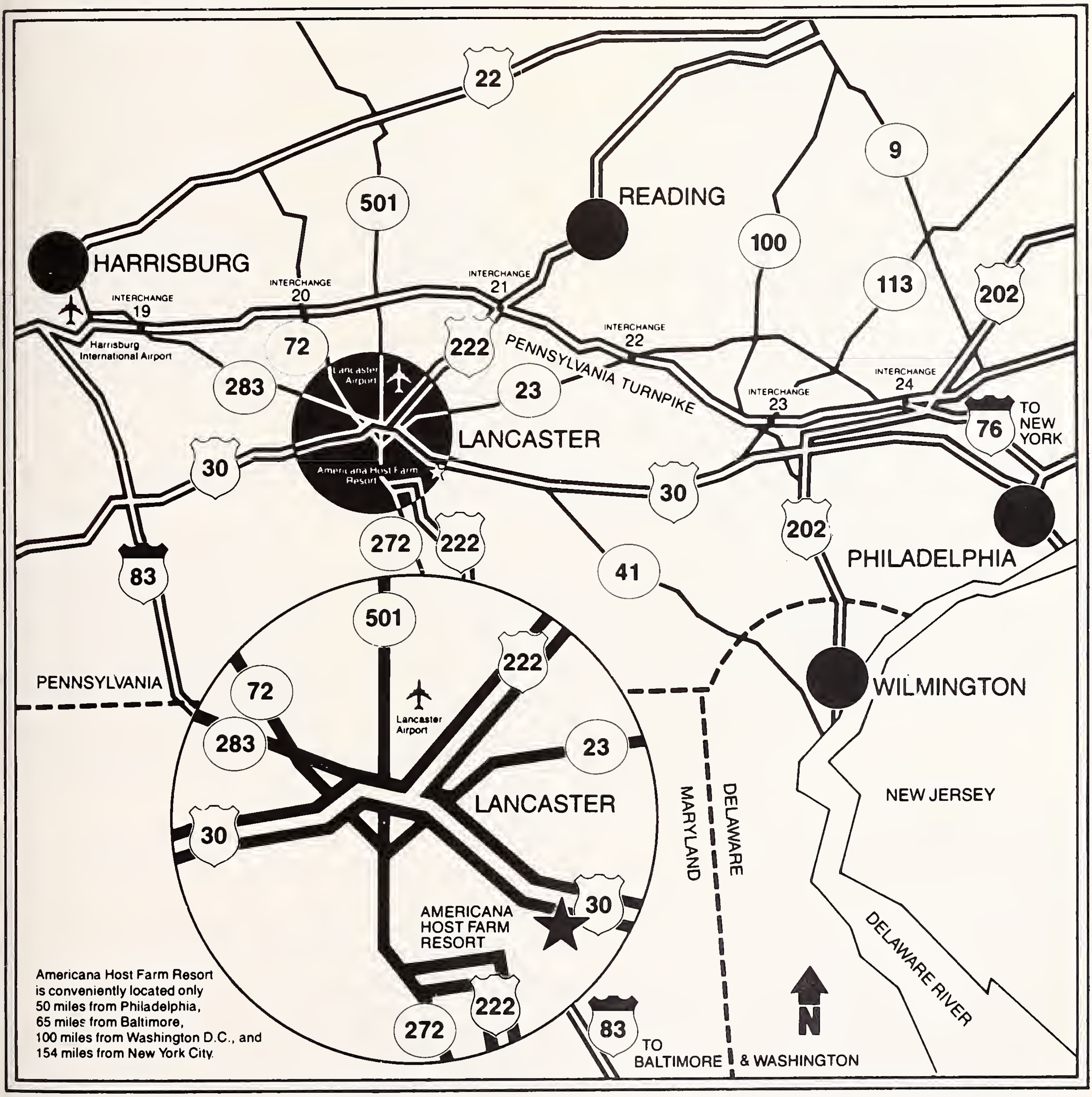
CHECK-IN: AFTER 4 PM
CHECK-OUT: BY 12 NOON

MEDICAL SOCIETY OF NEW JERSEY—220TH ANNUAL MEETING

LOCATION MAP

Americana
Host Farm Resort

2300 Lincoln Highway East (Route 30), Lancaster, Pennsylvania 17602 (717) 299-5500



Americana Host Farm Resort is conveniently located only 50 miles from Philadelphia, 65 miles from Baltimore, 100 miles from Washington D.C., and 154 miles from New York City.

FROM NEW YORK

Take New Jersey Turnpike South to Pa. Turnpike. Take Pa. Turnpike West to Interchange No. 21. Travel South on U.S. Rte. 222 to U.S. Route 30. Travel East on U.S. Route 30, approximately 5 miles to Americana Host Farm Resort.

FROM PITTSBURGH & POINTS WEST

Travel East on Pa. Turnpike to Int. 19. Go South on I-283 for 30 miles into Route 30 East for 6 miles to Americana Host Farm Resort.

FROM PHILADELPHIA

Take Pa. Turnpike West to Interchange No. 21. Travel South on U.S. Rte. 222 to U.S. Route 30. Travel East on U.S. Route 30, approximately 5 miles to Americana Host Farm Resort.

FROM BALTIMORE & WASHINGTON

Go North on Interstate Route 83 to York, Pa. Turn East on U.S. Route 30 for 32 miles to Americana Host Farm Resort.

BY AIR

Harrisburg International Airport (just 35 minutes from Host by limousine or car) via TWA and U.S. Air, and Lancaster Airport only minutes away with daily flights via U.S. Air.

BY RAILROAD

Amtrak to Lancaster Station. Taxi and Rent-a-Car Service available.

The Academy of Medicine of New Jersey
Community Memorial Hospital
Kimball Medical Center
St. Michael's Medical Center

present
a

MAJOR SYMPOSIUM ON INTERESTING PROBLEMS IN INFECTIOUS DISEASES

on

WEDNESDAY, APRIL 16, 1986
3:00 P.M.-8:00 P.M.

at

Palms Restaurant Lounge & Banquets
Lakewood, New Jersey

Following this program participants will be able to:

- —Discuss developments in recognition and diagnosing infection with emphasis on pneumonia, UTI, skin and soft tissue disease.
- —Outline office recognition, diagnosis, and management of infectious diseases in the office with emphasis on STD's and antibiotics.
- —Identify selected newer infectious diseases and associated laboratory studies for viral infections, legionella, chlamydia.
- —Discuss antibiotic usage for management of new infectious diseases.

For further information contact:

Deborah Gray
EXECUTIVE OFFICES
The Academy of Medicine of New Jersey
Two Princess Road
Lawrenceville, New Jersey 08648
(609) 896-1717

REGISTRATION

INTERESTING PROBLEMS IN INFECTIOUS DISEASES

Wednesday, April 16, 1986
Palms Restaurant Lounge & Banquets
Lakewood, New Jersey

CHECK ONE BELOW: (Registration includes dinner and refreshments)

___ AMNJ Fellows ($20.00)
___ Non-Member Physicians ($25.00)
___ Residents and Medical Students ($10.00)

Please make checks payable to:
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and mail to: Two Princess Road
Lawrenceville, New Jersey 08648

I enclose $____ for ____ Resevation(s). (Additional names listed separately)

NAME (please print) ____________________

ADDRESS ____________________

NO REFUNDS AFTER PROGRAM IS HELD

The Academy of Medicine of New Jersey
The Medical Society of New Jersey
The N.J. State Department of Health

present a

SYMPOSIUM ON AIDS AND THE IV DRUG ABUSER

on

WEDNESDAY, APRIL 16, 1986
8:30 A.M.-3:00 P.M.

at

MSNJ HEADQUARTERS
LAWRENCEVILLE, NEW JERSEY

Following this program participants will be able to:

- —Identify epidemiologic problems with AIDS and the uniqueness of New Jersey's AIDS population.
- —Discuss public policy issues including ethical delimmas among selected populations including children, prisoners, people with AIDS and family members.
- —Recognize uses and weaknesses of current HTLV3 antibody testing.
- —Outline unique diagnostic issues for people with AIDS from three perspectives; hospitals, residential treatment facilities and hospices.

For further information contact:

Deborah Gray
EXECUTIVE OFFICES
The Academy of Medicine of New Jersey
Two Princess Road
Lawrenceville, New Jersey 08648
(609) 896-1717

The Academy of Medicine
of New Jersey
&
New Jersey Orthopaedic Society

present

11th ANNUAL NEW JERSEY ORTHOPAEDIC SYMPOSIUM

on

SATURDAY, APRIL 5, 1986
8:00 a.m.-4:30 p.m.

UMDNJ-Rutgers Medical School
Piscataway, New Jersey

This Spring Symposium is designed to provide an opportunity for Orthopaedists in the tri-state area to hear timely topics of current interest presented by national leaders in these fields. In addition, members of the New Jersey Orthopaedic Society will present papers on their areas of social interest.

For further information contact:

Deborah Gray
EXECUTIVE OFFICES
The Academy of Medicine
of New Jersey
Two Princes Road
Lawrenceville, New Jersey 08648
(609) 896-1717

CME CALENDAR

The following is a list of continuing medical education courses for the next two months. Contact the sponsoring organization for further information.

This list is compiled through the cooperation of the Committee on Medical Education of the Medical Society of New Jersey, The Academy of Medicine of New Jersey, the New Jersey Chapter of the American Academy of Family Physicians, and the Office of Continuing Medical Education of the UMDNJ. For information on accreditation, please contact the sponsoring organization(s), indicated by italics—last line of each item.

ANESTHESIOLOGY

April

1 **Anesthesia Lectures**
8 4 P.M.—Medical Science Bldg.,
15 E-545, NJ Medical School, Newark
23 *(UMDNJ)*
29

7 **Anesthesia Lectures**
14 7 A.M.—Medical Science Bldg.,
21 E-545, NJ Medical School, Newark
28 *(UMDNJ)*

7 **Anesthesia Lectures**
14 7:45 A.M.—Auditorium, 3rd Fl.,
21 Newark Beth Israel Medical Center,
28 Newark
(UMDNJ)

23 **Anesthesia Grand Rounds**
4-5 P.M.—University Hospital,
H-350, Newark
(UMDNJ)

May

5 **Anesthesia Lectures**
12 7 A.M.—Medical Science Bldg.,
19 E-545, NJ Medical School, Newark
(UMDNJ)

5 **Anesthesia Lectures**
12 7:45 A.M.—Auditorium, 3rd Fl.,
19 Newark Beth Israel Medical Center
(UMDNJ)

6 **Anesthesia Lectures**
13 4 P.M.—Medical Science Bldg.,
20 E-545, NJ Medical School, Newark
27 *(UMDNJ)*

28 **Anesthesia Grand Rounds**
4-5 P.M.—University Hospital,
H-350, Newark
(UMDNJ)

CARDIOLOGY

April

25 **Cardiothoracic Journal Club**
7:30 P.M.—13 Ridgeway Drive, West Orange
(UMDNJ)

May

30 **Cardiothoracic Journal Club**
7:30 P.M.—13 Ridgeway Drive, West Orange
(UMDNJ)

MEDICINE

April

Mini-Residency in Proctology and Diseases of the Colon
One Week Commitment—contact Overlook Hospital, Summit
(Overlook Hospital and AMNJ)

1 **Pulmonary Conference**
8 8-9 A.M.—University Hospital,
15 Newark
22 *(UMDNJ)*
29

2 **Rehabilitation Medicine**
9 4:15-5:15 P.M.—Kessler Institute,
16 West Orange
23 *(UMDNJ)*
30

2 **Mini-Residency in Occupational**
18 **Medicine**
8:30 A.M.-4 P.M.—Contact UMDNJ-Rutgers Medical School,
Piscataway
(UMDNJ and AMNJ)

2 **Common Skin Problems**
1:30-2:30 P.M.—Rutgers Community Health Plan, 57 U.S. Hwy #1, New Brunswick
(Rutgers Community Health Plan and AMNJ)

2 **Heart Failure**
9 **Diseases of the Pancreas**
16 **Inflammatory Bowel Disease**
23 **Principles of Drug Therapy and**
30 **Drug Interaction**
Bradyarrhythmia
8:30-10 A.M.—Alexian Brothers Hospital, Elizabeth
(Alexian Brothers Hospital and AMNJ)

2 **Medical Lecture Series**
9 10:30 A.M.-12 noon—Christ
16 Hospital, Jersey City
23 *(Christ Hospital and AMNJ)*
30

3 **Human Genetics**
10 4-6 P.M.—Institute for Medical
17 Research, Copewood St., Camden
24 *(Institute for Medical Research and AMNJ)*

3 **Trauma Conference**
10 7:45-8:45 A.M.—University Hospital,
17 B-349, Newark
24 *(UMDNJ)*

3 **Digestive Disease Case Report**
10 8-9 A.M.—Medical Science Bldg.,
17 H-249, NJ Medical School, Newark
24 *(UMDNJ)*

3 **Digestive Disease Research and**
10 **Literature Review Conference**
17 9-10 A.M.—Medical Science Bldg.,
24 H-249, NJ Medical School, Newark
(UMDNJ)

3 **Allergy/Immunology Lecture**
10 11 A.M.-12 noon—United Hospital,
17 Newark
24 *(UMDNJ)*

4 **Dept. of Medicine Grand Rounds**
11 12 noon-1 P.M.—Medical Science
18 Bldg., B-610, NJ Medical School,
25 Newark
(UMDNJ)

4 **Major Advances in Cancer—1986**
8 A.M.-4 P.M.—NJ Medical School, Piscataway
(UMDNJ and AMNJ)

4 **Cancer Research Colloquium**
11 12 noon-1 P.M.—Medical Science
18 Bldg., G-506, NJ Medical School,
25 Newark
(UMDNJ)

4 **Urology Basic Surgery Conference**
11 2-3 P.M.—Medical Science Bldg.,
18 C-600, Newark
25 *(UMDNJ)*

4 **Urology Grand Rounds**
11 3-5 P.M.—Medical Science Bldg.,
18 C-600, Newark
25 *(UMDNJ)*

5 **Nutrition Support of Trauma Patients**
8 A.M.-4 P.M.—Marriott Hotel, Newark Airport
(UMDNJ and AMNJ)

7 **Tumor Conference**
14 8-9 A.M.—University Hospital,
21 H-349, Newark
28 *(UMDNJ)*

8 **Advances in Allergy and Immunology 1986**
8:30-1:30 P.M.—Holy Name Hospital, Teaneck
(Holy Name Hospital and AMNJ)

10 **State of the Art: E-B Virus**
11 A.M.—St. Joseph's Hospital and Medical Center, Paterson
(St. Joseph's Hospital and Medical Center)

11 **Clinical Concepts in Rheumatology/Immunology**
12:15-1:15 P.M.—Kennedy Memorial Hospital, Stratford
(UMDNJ)

12 **Advances in Clinical Andrology**
8:15 A.M.-4:15 P.M.—Rutgers Medical School, Piscataway
(UMDNJ)

15 **IVP Conference**
5-8 P.M.—East Orange VA Hospital, Rm. 142
(UMDNJ)

16 **AIDS and the IV Drug Abuser**
9 A.M.-4 P.M.—MSNJ Executive Offices, Lawrenceville
(NJ State Dept. of Health, MSNJ, and AMNJ)

16 **Dermatological Conference**
6-9 P.M.—Rutgers Community

PRIMARY CARE
INTERNAL MEDICINE

Diagnostic and
Management Strategies

May 7-9, 1986

Cancer Screening • Coagulation Disorders • Hepatitis • Ischemic Heart Disease • Obstructive Airway Disease • Peptic Ulcer Disease • Renal Failure • Venous Disease • Diagnostic Strategies • Acute/Chronic Therapeutic • Rationals • Cost Effectiveness

Jefferson
Medical
College
of Thomas Jefferson University
Philadelphia, PA 19107

For information regarding registration call:
Office of Continuing Medical Education
(215) 928-6992

June 21, 1986

6TH ANNUAL ADVANCES IN GASTROENTEROLOGY

Bally's Park Place
Atlantic City, New Jersey

Sponsored by the Gastrointestinal Section of the Hospital of the University of Pennsylvania and the Continuin Medical Education Department of the Underwoo Memorial Hospital, Woodbury, New Jersey.

Category 1 credit offered

INFORMATION: Registration Supervisor, SLACK Incorporated, 6900 Grove Road, Thorofare, New Jerse 08086, 609-848-1000.

LIKOFF CARDIOVASCULAR INSTITUTE
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CARDIOLOGY UPDATE. . .

IS DESIGNED FOR THE PHYSICIAN AND PROVIDES AN INTENSIVE SURVEY OF THE CURRENT STATUS OF CLINICAL CARDIOLOGY. . .

WEDNESDAY, APRIL 2, 1986

AORTIC VALVE DISEASE

MODERATOR: MARIELL J. LIKOFF, M.D.

3:00	Case Presentation: Aortic Stenosis	*Bruno Manno, M.D.*
3:30	Discussion, Including Work-Up and Management	*Ronald S. Pennock, M.D.*
4:00	Case Presentation: Aortic Regurgitation	*Daniel Mason, M.D.*
4:30	Discussion, Including Work-Up and Management	*Stanley Spitzer, M.D.*
5:00	Questions and Answers	*The Audience*

LECTURE HALL "A"—2nd floor New College Building, Hahnemann University
15th and Vine Streets, Philadelphia, PA
• NO REGISTRATION FEE • NO ADVANCE REGISTRATION REQUIRED •
• CME CATEGORY I CREDITS CERTIFIED •
WINE & CHEESE SERVED FOLLOWING CONFERENCE

Health Plan, 57 U.S. Hwy #1, New Brunswick
(UMDNJ and AMNJ)

16 **Inter-hospital Rheumatology Rounds**
4:30-6:30 P.M.—University Hospital, I-355, Newark
(UMDNJ-NJ Medical School)

16 **Interesting Cases in Infectious Diseases**
3-7 P.M.—Palms Restaurant, Lakewood
(AMNJ)

16 **Grand Rounds in Ophthalmology**
7-9 P.M.—United Hospitals Annex Bldg, Newark
(UMDNJ)

17 **Current Concepts in the Prevention and Management of Osteoporsis**
5-6:30 P.M.—Somerset Medical Center, Somerville
(Somerset Medical Center and AMNJ)

19 **Urological Emergencies in the Newborn**
26 Role and Operation of Day Surgeon Unit in a Tertiary Care Center
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

May

Mini-Residency in Proctology and Diseases of the Colon
One Week Commitment—Contact Overlook Hospital, Summit
(Overlook Hospital and AMNJ)

1 **Human Genetics**
8 4-6 P.M.—Institute for Medical Research, Copewood St., Camden
(Institute for Medical Research and AMNJ)

1 **Trauma Conference**
8 7:45-8:45 A.M.—University Hospital,
15 B-349, Newark
22 *(UMDNJ)*
29

1 **Digestive Disease Case Report**
8 8-9 A.M.—Medical Science Bldg.,
15 H-249, NJ Medical School, Newark
22 *(UMDNJ)*
29

1 **Digestive Disease Research and**
8 **Literature Review Conference**
15 9-10 A.M.—Medical Science Bldg.,
22 H-249, NJ Medical School, Newark
29 *(UMDNJ)*

1 **Allergy/Immunology Lecture**
8 11 A.M.-12 noon—United Hospital,
15 Newark
22 *(UMDNJ)*
29

2 **Dept. of Medicine Grand Rounds**
9 12 noon-1 P.M.—Medical Science
16 Bldg., B-610, NJ Medical School,
23 Newark
30 *(UMDNJ)*

2 **Cancer Research Colloquium**
9 12 noon-1 P.M.—Medical Science
16 Bldg., G-506, NJ Medical School,
23 Newark
30 *(UMDNJ)*

2 **Urology Basic Surgery Conference**
9 2-3 P.M.—Medical Science Bldg.,
16 C-600, Newark
23 *(UMDNJ)*
30

2 **Urology Grand Rounds**
9 3-4 P.M.—Medical Science Bldg.,
16 C-600, Newark
23 *(UMDNJ)*
39 **Tumor Conference**
12 8-9 A.M.—University Hospital,
19 Newark
(UMDNJ)

3 **Trauma**
8:45 A.M.-1 P.M.—St. Barnabas Medical Center, Livingston
(Saint Barnabas Medical Center)

6 **Pulmonary Conference**
13 8-9 A.M.—University Hospital,
20 Newark
27 *(UMDNJ)*

7 **Chronic Renal Failure**
14 **New Drugs in Hypertension**
8:30-10 A.M.—Alexian Brothers Hospital, Elizabeth
(Alexian Brothers Hospital and AMNJ)

7 **Medical Lecture Series**
14 10:30 A.M.-12 noon—Christ
21 Hospital, Jersey City
28 *(Christ Hospital and AMNJ)*

7 **Rehabilitation Medicine**
14 4:15-5:15 P.M.—Kessler Institute,
21 West Orange
28 *(UMDNJ)*

9 **Clinical Concepts in Rheumatology Immunology**
12:15-1:15 P.M.—Kennedy Memorial Hospital, Stratford
(UMDNJ)

15 **Adjuvant Chemohormonal Therapy in Breast Cancer**
5-6:30 P.M.—Somerset Medical Center, Somerville
(Somerset Medical Center and AMNJ)

21 **Inter-hospital Rheumatology Rounds**
4:30-6:30 P.M.—University Hospital, I-355, Newark
(UMDNJ)

21 **Dermatological Conference**
6-9 P.M.—Rutgers Community Health Plan, 57 U.S. Hwy. #1, New Brunswick
(UMDNJ-Rutgers Medical School and AMNJ)

20 **IVP Conferences**
5-8 P.M.—East Orange VA Hospital, Rm. 142
(UMDNJ)

21 **Patterns of Cocaine Use**
1:30-2:30 P.M.—Rutgers Community Health Plan, 57 U.S. Hwy #1, New Brunswick
(Rutgers Community Health Plan and AMNJ)

21 **Grand Rounds in Ophthalmology**
7-9 P.M.—United Hospitals Annex Bldg, Newark
(UMDNJ)

29 **Treatment of Herpes Diseases in Immunocompromised Patients**
11 A.M.-12 noon—St. Joseph's Hospital and Medical Center, Paterson
(St. Joseph's Hospital and Medical Center)

NEUROLOGY/PSYCHIATRY

April

1 **Abdicating Fathers, Homosexual Sons**
8:30-10:30 P.M.—Marriott Hotel, Saddle Brook
(NJ Council of Child and Adolescent Psychiatry and AMNJ)

2 **Grand Rounds in Psychiatry**
10:30 A.M.-12 P.M.—Medical Science Bldg., B-610, NJ Medical School, Newark
(UMDNJ)

3 **Case Seminars**
17 8-10 P.M.—312 Harding Drive, South Orange
(Advanced Psychiatric Study Group and AMNJ)

7 **Violence in a Marriage**
8:15-10:15 P.M.—39 Crescent Ave., Passaic
(Essex Psychiatric Seminar and AMNJ)

16 **Psychiatry Lecture**
10:30 A.M.-12 P.M.—Medical Science Bldg., B-610, NJ Medical School,

ARE YOU MOVING?

If so, please send a change of address to *NEW JERSEY MEDICINE,* Medical Society of New Jersey, Two Princess Road, Lawrenceville, NJ 08648, at least six weeks before you move.

Name ____________________

Address ____________________

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June 9-13, 1986

Update Your Medicine 1986. Association of Practicing Physicians of The New York Hospital and Cornell University Medical College. 37 hours Category I credit, (42½ if optional workshops taken) a one week review of all subspecialities of internal medicine. 9 major review lectures, 16 workshops, 2 Meet the Professor luncheons, optional practical workshops in breast/pelvic and male genitorectal examinations. Held at The New York Hospital-Cornell, 1300 York Avenue at 69th Street, New York, Information: Office of CME, 212-472-6119. Dr. Lila A. Wallis is Course Director. Also call us for information about Update Your Medicine Ithaca course July 14-18, 1986.

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Newark
(UMDNJ)

6 **Psychiatric Lecture**
1-3 P.M.—Essex County Hospital Center, Cedar Grove
(UMDNJ)

May

1 **Case Seminars**
15 8-10 P.M.—312 Harding Drive, South Orange
(Advanced Psychiatric Study Group and AMNJ)

5 **A Delayed Reaction to Rape**
8:15-10:15 P.M.—543 Park Street, Upper Montclair
(Essex Psychiatric Seminar and AMNJ)

7 **Grand Rounds in Psychiatry**
10:30 A.M.-12:30 P.M.—Medical Science Bldg., B-610, NJ Medical School, Newark
(UMDNJ)

21 **Psychiatry Lecture**
10:30 A.M.-12:30 P.M.—Medical Science Bldg., B-610, NJ Medical School, Newark
(UMDNJ)

21 **Psychiatric Lecture**
1-3 P.M.—Essex County Hospital Center, Cedar Grove
(UMDNJ)

24 **Behavioral Medicine**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

OBSTETRICS/GYNECOLOGY

April

1 **Pathology/Ob/Gyn Conference**
8 5:15-6:15 P.M.—Medical Science
15 Bldg., C-579, NJ Medical School,
22 Newark
29 *(UMDNJ)*

May

6 **Pathology/Ob/Gyn Conference**
13 5:15-6:15 P.M.—Medical Science
20 Bldg., C-579, NJ Medical School,
27 Newark
(UMDNJ)

PATHOLOGY

April

1 **Anatomic Pathology**
8 5:15 P.M.—Medical Science Bldg.,
15 C-555, NJ Medical School, Newark
22 *(UMDNJ)*
29

1 **Pathology/Ob/Gyn Conference**
8 5:15-6:15 P.M.—Medical Science
15 Bldg., C-579, NJ Medical School,
22 Newark
29 *(UMDNJ)*

2 **Neuropathology Conference**
16 10:30-12:30 P.M.—Medical Science Bldg., C-555, NJ Medical School, Newark
(UMDNJ)

2 **Pathology Grand Rounds**
9 1-5 P.M.—Medical Science Bldg.,
16 C-555, NJ Medical School
23 *(UMDNJ)*
30

3 **Pathology/Radiology/Orthopedics**
10 **Conference**
17 8-9 A.M.—University Hospital,
24 Newark
(UMDNJ)

3 **Pathology Experimental Seminar**
10 12 noon-1 P.M.—Medical Science
17 Bldg., C-555, NJ Medical School,
24 Newark
(UMDNJ)

9 **Pathology Experimental Seminar**
23 12 noon-1 P.M.—Medical Science Bldg., C-555, Newark
(UMDNJ)

May

1 **Pathology/Radiology/Orthopedics**
8 **Conference**
15 8-9 A.M.—University Hospital,
22 Newark
29 *(UMDNJ)*

1 **Pathology Experimental Seminar**
8 12 noon-1 P.M.—Medical Science
15 Bldg., C-555, NJ Medical School,
22 Newark
29 *(UMDNJ)*

6 **Anatomic Pathology**
13 5:15 P.M.—Medical Science Bldg.,
20 C-555, NJ Medical School, Newark
27 *(UMDNJ)*

6 **Pathology/Ob/Gyn Conference**
13 5:15-6:15 P.M.—Medical Science
20 Bldg., C-579, NJ Medical School,
27 Newark
(UMDNJ)

7 **Neuropathology Conference**
21 10:30-12:30 P.M.—Medical Science Bldg., C-555, NJ Medical School, Newark
(UMDNJ)

7 **Pathology Experimental Seminar**
21 12 noon-1 P.M.—Medical Science Bldg., C-555, NJ Medical School, Newark
(UMDNJ)

7 **Pathology Grand Rounds**
14 1-5 P.M.—Medical Science Bldg.,
21 C-555, NJ Medical School, Newark
28 *(UMDNJ)*

PEDIATRICS

April

4 **Advances in Pediatrics**
11 9:30-10:30 A.M.—Medical Science
18 Bldg., B-610, NJ Medical School,
25 Newark
(UMDNJ)

9 **Urologic Emergencies in the Newborn**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

May

2 **Advances in Pediatrics**
9 9:30-10:30 A.M.—Medical Science
16 Bldg., B-610, NJ Medical School,
23 Newark
30 *(UMDNJ)*

RADIOLOGY

April

2 **Radiology Grand Rounds**
16 2-4 P.M.—University Hospital, H-346, Newark
(UMDNJ)

3 **Pathology/Radiology/Orthopedics**
10 **Conference**
17 8-9 A.M.—University Hospital,
24 Newark
(UMDNJ)

May

1 **Pathology/Radiology/Orthopedics**
8 **Conference**
15 8-9 A.M.—University Hospital,
22 Newark
29 *(UMDNJ)*

7 **Radiology Grand Rounds**
21 2-4 P.M.—University Hospital, H-346, Newark
(UMDNJ)

SURGERY

April

1 **Surgical Grand Rounds**
8 7-9 A.M.—Hackensack Medical
15 Center
29 *(Hackensack Medical Center and AMNJ)*

5 **Surgical Treatment of Cardiothoracic Diseases**
10 A.M.-12 noon—Medical Science Bldg., G-506, NJ Medical School, Newark
(UMDNJ)

5 **Surgical Morbidity and Mortality**
12 **Conference**
19 8:30-9:30 A.M.—Medical Science
26 Bldg., G-506, Newark
(UMDNJ)

7 **Tumor Conference**
14 8-9 A.M.—University Hospital,
21 H-346, Newark
28 *(UMDNJ)*

7 **Surgical Grand Rounds and**
14 **Lecture Series**
21 4:30-6:30 P.M.—Medical Science
28 Bldg., B-610, NJ Medical School, Newark
(UMDNJ)

22 **Neoplastic Disease of the Lung**
8-10 P.M.—Englewood Club, 115 E. Palisade Ave., Englewood
(Englewood Surgical Society and AMNJ)

26 **Role and Operation of a Day Surgical Unit in a Tertiary Care Center**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital

3 **Surgical Treatment of Cardiothoracic Diseases**
10 A.M.-12 noon—Medical Science Bldg., G-506, NJ Medical School, Newark
(UMDNJ)

3 **Surgical Morbidity and Mortality**
10 **Conference**
17 8:30-9:30 A.M.—Medical Science
24 Bldg., G-506, Newark
(UMDNJ)

5 **Tumor Conference**
12 8-9 A.M.—University Hospital,
19 Newark
(UMDNJ)

5 **Surgical Grand Rounds and**
12 **Lecture Series**
19 4:30-6:30 P.M.—Medical Science

Bldg., B-610, NJ Medical School,
Newark
(UMDNJ)

6 **Surgical Grand Rounds**
7-9 A.M.—Hackensack Medical
Center
*(Hackensack Medical Center and
AMNJ)*

27 **Pediatric Surgery-Ambiguous
Genitalia**
8-10 P.M.—Englewood Club, 115 E.
Palisade Ave., Englewood
*(Englewood Surgical Society and
AMNJ)*

10 **Microsurgery
(Neurovascular—Tendon)**
8 A.M.—Newcomb Hospital,
Vineland
(Newcomb Hospital)

SURGICAL SPECIALTIES

April

3 **Pathology/Radiology/Orthopedics**
10 **Conference**
17 8-9 A.M.—University Hospital,
24 Newark
(UMDNJ)

Orthopedic Symposium
4 3:15-5:30 P.M.—Case
Presentations, Marriott,
Somverville
5 7:30 A.M.-4 P.M.—Rutgers Medical
School, Piscataway
(UMDNJ and AMNJ)

7 **Orthopedic Grand Rounds**
14 6-8 P.M.—Medical Science Bldg.,
21 B-556, NJ Medical School, Newark
28 *(UMDNJ)*

10 **Neurosurgery Lecture**
4-6 P.M.—University Hospital,
E-343, Newark
(UMDNJ)

May **Pathology/Radiology/Orthopedics**
1 **Conference**
8 8-9 A.M.—University Hospital,
15 Newark
22 *(UMDNJ)*

5 **Orthopedic Grand Rounds**
12 6-8 P.M.—Medical Science Bldg.,
19 B-556, NJ Medical School, Newark
(UMDNJ)

15 **Neurosurgery Lecture**
4-6 P.M.—University Hospital,
E-343, Newark
(UMDNJ and AMNJ)

MISCELLANEOUS

April

9 **Office Management of Common
Medical Problems**
12 noon-4 P.M.—Kennedy Memoria
Hospital, Stratford
(UMDNJ)

May

14 **Office Management of Common
Medical Problems**
12 noon-4 P.M.—Kennedy Memoria
Hospital, Stratford
(UMDNJ)

Common Eye Diseases and Their Management; Manual of Paediatric Gastroenterology; 1985 Yearbook of Emergency Medicine; Psychological Aspects of Cancer

Common Eye Diseases and Their Management

N.R. Galloway. New York, NY, Springer-Verlag, 1985. Pp. 278. Illustrated. ($35)

Common Eye Diseases and Their Management was written primarily for medical students and nonspecialist postgraduates. Stimulated by a phenomenon in Great Britain where hospital waiting lists for eye care problems are extensive and medical student exposure to ophthalmology is scant, Dr. Galloway has provided us with a concise, easy-to-read textbook. While this basically clinical and practical book is not intended as a surgical manual, some of the simpler surgical techniques, which easily could be performed by the general practitioner, are mentioned.

This book, with five sections, introduces the subject of ophthalmology with a brief overview of the scope and history of the field, pointing out that this specialty boasts more clinical and scientific journals than any other medical specialty. The basic structure of the human eye is simply and comprehensively covered, followed by "what-is" and "how-to's" explaining standard tests and examination procedures, e.g. "what-is" visual acuity and "how-to" use an ophthalmoscope. The following section is on primary eye care problems and is geared to the nonophthalmologist including many problems that can be handled by knowledgeable general practitioners. Included in this section are common diseases of the eyelids (watering eye, dry eye, eyelid deformities, lid tumors, and lid injuries), conjunctiva and cornea (subconjunctival hemorrhage, conjunctivitis, corneal foreign bodies, corneal ulceration, corneal degeneration, corneal edema, and absent corneal sensation), red eye, failing vision, headache, and contact lenses. An entire section also is devoted to problems for the eye surgeon (cataract, glaucoma, retinal detachment, squint, tumors, and trauma) and another to problems for the medical ophthalmologist (inflammation, aging, pediatrics, systemic disease, neuro-ophthalmology, genetics, and pharmacology). The last section deals with blindness.

The author provides the reader with further reading at the end of each section of the book. One hundred nineteen figures punctuate this well-written, easily readable introduction to the field of eye care and ophthalmology. Dr. Galloway has more than achieved his goals.

Alfonse A. Cinotti, M.D.

Manual of Paediatric Gastroenterology

John H. Tripp and David C.A. Candy. New York, NY, Churchill Livingstone, 1985. Pp. 168. ($16.50)

This text is a useful manual—it is compact and can be carried in the pocket or stored easily on the shelf. The information provided is in précis form designed for the student, resident, pediatrician, or family physician, in order to orient that practitioner with respect to a particular clinical problem.

The book is not a thorough treatment of the general subject or any particular segment. Where the Tripp and Candy volume may be helpful is when the physician suspects a clinical problem for which he or she needs a start. The student or resident, for whom many types of disorders are new, is offered a chance to see where to go in organizing clinical care and in searching literature for more detailed information.

Occasional reference is made to brand-name products, but always those available in Great Britain, where the authors teach. As a result, a physician practicing in the United States may be hard put to know what is meant. The brief summaries in the final pages are just not sufficient to know what products sold in our country are comparable.

In summary, this is a useful volume when used for the purpose intended by the authors.

Avrum L. Katcher, M.D.

1985 Yearbook of Emergency Medicine

David K. Wagner, M.D., (ed). Chicago, IL, Year Book Medical Publishers, Inc., 1985. Pp. 408. Illustrated. ($42.95)

This yearbook consists of 408 pages containing 343 abstracts from 126 journals worldwide, but predominantly from journals published in the United States in 1983 and the first four months of 1984. The editorial board includes the instructors in emergency medicine at the University of Pennsylvania.

The categories covered in this book are as follows: acute systems pathophysiology; trauma injuries; organ system injuries; special injuries; emergency procedures; techniques and instrumentation; and emergency medical service systems.

After each abstract there is an interpretive analysis of the particular abstract—an excellent idea.

Emergency medicine is fairly well-documented, but incomplete. Many of the abstracts are the result of research work. Thus, the material presented may be used by the reader as a basis for other similar research.

This book is not one that could be used adequately for information in emergency medicine by a hospital emergency department or private physician; we have excellent texts in emergency medicine that are more complete and have sufficient bibliographical references of all emergency medical conditions after each chapter combining necessary subject matter and a bibliography for further study of that condition and at a more reasonable cost.

Jack R. Karel, M.D.

Psychological Aspects of Cancer

M. Watson and T. Morris (eds). Oxford, England, Pergamon Press, 1985. ($29.50)

This 100-page pamphlet represents a compilation of papers presented during a conference on the psychological aspects of cancer held in London in November 1983.

Evidence is accumulating supporting the contention that psychological factors may influence strongly the occurrence of cancer, the success of treatment, and even the duration of survival. Some researchers are finding that some patients survive longer if they show a "fighting spirit" while others who may be apathetic or give in to hopelessness or helplessness tend to show a shorter survival. Others have found that psychological factors may be involved in carcinogenesis or in promoting the risk of cancer. The mechanisms by which psychological factors could influence malignant neoplasms may be neuroendocrine and immunological but remain controversial.

The papers herein address three major areas pertaining to these issues. The first consists of three papers reviewing psychological interactions in cancer patients and the role of neuroendocrine transmitter substances as well as the role of stress and emotional responses. The second group of three papers deals with the role of psychological responses to a diagnosis of cancer and underscores the important role that psychiatrists and other psychosocial support personnel can have in aiding the clinical oncologist in patient management. The final two papers deal with the practical aspects of psychological intervention in patient management.

The concise but comprehensive volume should command the attention of all physicians involved in cancer care because it calls attention to important approaches to the cancer problem heretofore virtually ignored.

Alan J. Lippman, M.D.

Drs. Avella-Johnson; Baker; Barbash; Bernheisel; Brody; Felmlee; Fogel; Hinckley; Iger; Insolera; Kuchlewski; Ridley; Southard

Dr. C. Avella-Johnson

At the untimely age of 51, Carolyn Avella-Johnson, M.D., died at West ersey Hospital, Voorhees, on No-ember 13, 1985, after a two-month lness. Born in Chicago, Dr. Avella-ohnson received her medical egree from Temple University Medcal School in 1960, and for all of her brief career was affiliated with West ersey Hospital as resident anesthe-iologist. She was a member of our Camden County component, and of he American Medical Association. Dr. Avella-Johnson is survived by her husband, Donald H. Johnson, M.D., also an anesthesiologist, and member of our Society.

Dr. Elsworth F. Baker

A psychiatrist in Fair Haven, Elsworth Fredrick Baker, M.D., died on June 2, 1985, at the venerable age of 82. Born in Summit, South Dakota, Dr. Baker received his medical education at the University of Mani-oba Faculty of Medicine, Winnipeg, Canada, and was graduated in 1928. He was a member of our Monmouth County component, and of the American Medical Association. Dr. Baker was a Fellow of the American Psychiatric Association, and had been board certified in his specialty. n 1978, he was a recipient of MSNJ's Golden Merit Award, noting his 50 years as a physician.

Dr. Roslyn Barbash

A leader in the fight against air pollution, Roslyn Barbash, M.D., died on October 18, 1985, at the age of 74. Born in New York City, Dr. Barbash received her medical degree from Bellevue Hospital Medical College, New York, in 1934. She established an office in Teaneck, and for more than 35 years served that community as an allergist. In 1968, Dr. Barbash was appointed to the New Jersey State Department's Clean Air Council by Governor Hughes, and was reappointed by Governor Cahill in 1971. She took a leave of absence from her practice to devote full time to fighting air pollution. She had served as chairman of Bergen County's and MSNJ's Committees on Air Pollution Control and Environmental Health, and was a member of the Council on Public Health. Dr. Barbash was founder of the allergy clinic at Englewood Hospital and director of allergies at the Vanderbilt Clinic of Columbia-Presbyterian Medical Center, Manhattan. She also was affiliated with Bergen Pines County Hospital, Paramus. A member of our Bergen County component, Dr. Barbash was a recipient of MSNJ's Golden Merit Award, honoring her 50 years as a physician.

Dr. Louis E. Bernheisel

We just have learned of the death, on June 11, 1985, of Louis Edmond Bernheisel, M.D., a retired general practitioner from Tuckahoe. Born in 1900, Dr. Bernheisel received his medical degree from Hahnemann Medical College and Hospital, Philadelphia, in 1928. He was a member of our Cape May County component, and of the American Medical Association. Dr. Bernheisel had been affiliated with Shore Memorial Hospital, Somers Point, and with Burdette-Tomlin Medical Center, Cape May Court House. During World War II, he served with the medical corps for four years, emerging with the rank of lieutenant-colonel. Dr. Bernheisel received MSNJ's Golden Merit Award in 1978, honoring his 50th anniversary in medicine.

Dr. Michael Brody

A retired ophthalmologist, Michael Brody, M.D., died in San Diego, California, on October 12, 1985. Born in New York City, Dr. Brody received his medical degree from Cornell University Medical College, New York, in 1934. He established his practice in New Brunswick, later moving to East Brunswick. Dr. Brody was a member of our Middlesex County component and of the American Medical Association. He was board certified in his specialty, and a Fellow of the American College of Surgeons. Dr. Brody had been affiliated with Newark's Eye and Ear Infirmary, St. Peter's Medical Center, and Middlesex General Hospital, New Brunswick. During World War II, he served in the medical department of the United States Air Force. In 1984, Dr. Brody was a recipient of MSNJ's Golden Merit Award, honoring his 50 years as a physician.

Dr. John G. Felmlee

A radiologist in Bridgeton, John Gerald Felmlee, M.D., died on October 21, 1985. Born in 1922, in Dry Run, Pennsylvania, Dr. Felmlee was graduated from the University of Pennsylvania School of Medicine in 1950. He was a member of our Cumberland County component, and of the American Medical Association. Dr. Felmlee was board certified in his specialty, and had been affiliated with Bridgeton Hospital, after having served in medical facilities in Madison, West Virginia, and Lancaster and Altoona, Pennsylvania.

Dr. Richard L. Fogel

At the untimely age of 54, Richard L. Fogel, M.D., a Livingston endocrinologist, died in Saint Barnabas Medical Center, in that city on November 3, 1985. A native of Newark, Dr. Fogel received his medical degree from Chicago Medical School in 1957. He was a member of our Essex County component, was a Fellow of the American College of Physicians, and was board certified in his speciality. Dr. Fogel had been chief of endocrinology at Saint Barnabas Medical Center.

Dr. Livingston S. Hinckley

We have just learned of the death, after a prolonged illness, of Livingston Spraker Hinckley, II, M.D., in Florida, on September 21, 1985, at the remarkable age of 92. Dr.

Hinckley, a retired neuropsychiatrist, had been born in Newark, and received his medical degree from Jefferson Medical College in 1921. For 45 years he served the community of Newark, both in private practice, and through his affiliation with Newark City Hospital (now United Hospitals Medical Center). Dr. Hinckley had served the Veterans Administration Hospitals in Lyons and the Bronx, New York. He was a member of our Essex County component, and of the American Medical Association. In 1971, Dr. Hinckley was a recipient of MSNJ's Golden Merit Award, honoring his 50th year in medicine.

Dr. Jack Iger

The former chief of obstetrics and gynecology at East Orange General Hospital, Jack Iger, M.D., died at Saint Barnabas Medical Center, Livingston, on November 2, 1985, at only 59 years of age. Born in Vienna, Austria, Dr. Iger emigrated to this country and received his medical degree from Hahnemann Medical College and Hospital, Philadelphia, in 1952. He had been a member of our Essex County component and of the American Medical Association. Dr. Iger was board certified in his specialty, and a Fellow of the American College of Obstetricians and Gynecologists. He had been affiliated with Saint Barnabas Medical Center and the Hospital Center at Orange.

Dr. John A. Insolera

Belatedly we have learned of the death in Peoria, Arizona, on September 22, 1985, of John Anthony Insolera, M.D., an internist and rheumatologist. Born in Italy in 1918, Dr. Insolera emigrated to the United States and earned his medical degree at Long Island College of Medicine, New York, in 1944. He was board certified in his specialty, and was a member of our Passaic County component and of the American Medical Association. Prior to his move to Arizona, Dr. Insolera had been affiliated with St. Joseph's Hospital and Medical Center, Paterson. In recent years he had been affiliated with Boswell Hospital, Sun City; Valley View Hospital, Youngtown; and Thunderbird Hospital, Glendale; all in Arizona. During World War II, Dr. Insolera served in the medical corps of the United States Army for two years, emerging with the rank of captain.

Dr. Edward Kuchlewski

Just recently we were informed of the death on June 3, 1985, of Edward Kuchlewski, M.D., a retired general practitioner, who had practiced in Elizabeth for many years, prior to a move to Atlantic Highlands. A native of Poland, born in 1903, Dr. Kuchlewski received his medical degree from Ohio State University College of Medicine in 1931. He was a member of our Monmouth County component and of the American Medical Association. Dr. Kuchlewski had been affiliated with Saint Elizabeth and Alexian Brothers Hospitals, Elizabeth. In 1981, honoring his 50 years as a physician, MSNJ conferred its Golden Merit Award on Dr. Kuchlewski.

Dr. James B. Ridley

On October 26, 1985, cancer claimed the life of James Buchanan Ridley, M.D., retired surgeon and general practitioner, in Melbourne, Florida. A native of New York City, born in 1914, Dr. Ridley received his medical degree from Cornell University Medical College, New York, in 1941. He had served the community of Englewood for almost 30 years, and had been city physician for 15 years. Dr. Ridley was company physician for Prentice-Hall Publishing Company, Englewood Cliffs, for many years. He had been affiliated with Englewood Hospital and Bergen Pines County Hospital, Paramus. He was a member of our Bergen County component, was a Fellow of the American Society of Abdominal Surgeons, and was a Diplomate of the American Board of Abdominal Surgery. During World War II, Dr. Ridley served in the medical corps of the United States Army, attaining the rank of colonel.

Dr. Samuel C. Southard

After a three-year battle agains cancer, Samuel Casto Southard M.D., died on November 18, 1985, a Hahnemann University Hospital Philadelphia. A native of East Orange, born in 1922, Dr. Southard received his medical degree from Hahnemann Medical College in 1946. Immediately upon graduation he joined the medical corps of the United States Army in Korea where he served for three years. Upon his return to civilian life, Dr. Southard established a pediatric practice in Atlantic City, later switching to Ventnor where he continued unti his retirement in 1983. For three years, he was appointed director, by President Lyndon B. Johnson, of Task Force I, the National Commission on Product Safety. Some achievements of his efforts were safety caps on aspirin bottles, mandatory seatbelts in cars, and safety features on children's toys. Dr Southard also pioneered in the establishment of poison contro centers, and one of the first in the world, at Atlantic City Medica Center, was duplicated around the world. He was a member of our Atlantic County component, and of the American Medical Association, and had been board certified in his specialty. Dr. Southard also was an associate clinical professor of pediatrics at George Washington University School of Medicine, Washington D.C. He helped establish a free pediatric clinic at Atlantic City Medica Center, dedicated to providing medical care to the city's large minority population.

AUTHOR INFORMATION

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CONTENT

The educational content of each ssue appears as scientific articles, based on research, original concepts relative to epidemiology of disease, and treatment methodology; case reports based on unusual clinical experiences; review articles; clinical notes, succinct items on some aspect or new observation or technique of a case experience; and special articles, which include evaluations, policy and position papers, and reviews of nonscientific subjects. Other topics include commentary (critical narration); medical history; therapeutic drug information; pediatric briefs; nutrition update; and an opinion column. Editorials are prepared by the Editor and by guest contributors on timely and relevant subjects; editorials are the responsibility of the author. The Doctors' Notebook section contains organizational, informational, and administrative items from MSNJ and from the community. Letters to the Editor and book reviews are welcome and will be published as space permits. The principal aim in the preparation of a contribution should be relevance to diagnosis and treatment and to education of patients and professionals. Preference will be given to professional authors from New Jersey and to out-of-state lecturers who submit a suitable manuscript based on a presentation made in New Jersey.

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Submit two **manuscripts** that must be typewritten and double-spaced on 8½" by 11" paper. Statistical methods used in articles should be identified. Acknowledgements will be made only for specific preparation of an essential part of the manuscript.

Authors are asked to seek clarity, accuracy, and originality; attention to details of grammar, spelling, and typing are important.

The **title page** should include the full name, degrees, and affiliations of all authors, and the name and address of the author to whom reprint requests should be sent.

The author should submit a 40-word **abstract** to be used at the beginning of the article.

Tables must be typewritten and double-spaced on separate 8½" by 11" sheets, with a title and number. Symbols for units should be confined to column headings, and abbreviations, properly explained, should be kept to a minimum.

Illustrations should be professional quality, black-and-white glossy prints. The name of the author, figure number, and the top of the figure should be noted on a label attached to the back of each illustration. Where photographs of patients are used, the subjects should not be identifiable or publication permission, signed by the subject or responsible person, must be included with the photograph. Material taken from other publications must give credit to the source; written permission for republication from the original publisher must be submitted. The cost of color photographs must be borne by the author.

Generic names should be used with proprietary names indicated parenthetically or as a footnote with the first use of the generic name. Proprietary names of devices should be indicated by the registration symbol—®.

The **summary** of the article should not exceed 250 words; it should contain only essential facts.

References should not exceed 35 citations except in review articles, and should be cited consecutively in the text by numbers in parentheses at the end of the sentence. The reference list should be typewritten and double-spaced on separate 8½" by 11" sheets in the numerical order in which they are first cited in the text. The style of reference is that of *Index Medicus:*

1. Goldwyn RM: Subcutaneous mastectomy. *J Med Soc NJ* 74:1050-1052, 1977.
2. Dixon WJ, Massey FJ: *Introduction to Statistical Analysis.* New York, NY, McGraw-Hill, 1969, pp. 42-48.

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Receipt of each manuscript will be acknowledged and a copy delivered to the Editor who refers the paper to one or more members of the Editorial Board. The final decision is reserved for the Editor. No direct contact between the reviewers and the authors will be permitted, but authors will be informed of the reviewers' comments. The publication lag for original articles may be six months or more. Galley proofs will be submitted to the author for correction of typographical errors.

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WARNINGS: FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS.

BACTRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION. Clinical signs, such as rash, sore throat, fever, pallor, purpura or jaundice, may be early indications of serious reactions. In rare instances a skin rash may be followed by more severe reactions, such as Stevens-Johnson syndrome, toxic epidermal necrolysis, hepatic necrosis or serious blood disorder. Perform complete blood counts frequently.

BACTRIM SHOULD NOT BE USED IN THE TREATMENT OF STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have a greater incidence of bacteriologic failure when treated with Bactrim than with penicillin.

PRECAUTIONS:

General: Give with caution to patients with impaired renal or hepatic function, possible folate deficiency and severe allergy or bronchial asthma. In glucose-6-phosphate dehydrogenase-deficient individuals, hemolysis may occur, frequently dose-related.

Information for Patients: Instruct patients to maintain adequate fluid intake to prevent crystalluria and stone formation.

Laboratory Tests: Perform complete blood counts frequently; if a significant reduction in the count of any formed blood element is noted, discontinue Bactrim. Perform urinalyses with careful microscopic examination and renal function tests during therapy, particularly for patients with impaired renal function.

Drug Interactions: In elderly patients concurrently receiving certain diuretics, primarily thiazides, an increased incidence of thrombocytopenia with purpura has been reported. Bactrim may prolong the prothrombin time in patients who are receiving the anticoagulant warfarin. Keep this in mind when Bactrim is given to patients already on anticoagulant therapy and reassess coagulation time. Bactrim may inhibit the hepatic metabolism of phenytoin. Given at a common clinical dosage, it increased the phenytoin half-life by 39% and decreased the phenytoin metabolic clearance rate by 27%. When giving these drugs concurrently, be alert for possible excessive phenytoin effect. Sulfonamides can displace methotrexate from plasma protein binding sites, thus increasing free methotrexate concentrations.

Drug/Laboratory Test Interactions: Bactrim, specifically the trimethoprim component, can interfere with a serum methotrexate assay as determined by the competitive binding protein technique (CBPA) when a bacterial dihydrofolate reductase is used as the binding protein. No interference occurs if methotrexate is measured by a radioimmunoassay (RIA). The presence of trimethoprim and sulfamethoxazole may also interfere with the Jaffé alkaline picrate reaction assay for creatinine, resulting in overestimations of about 10% in the range of normal values.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Carcinogenesis: Long-term studies in animals to evaluate carcinogenic potential not conducted with Bactrim. *Mutagenesis:* Bacterial mutagenic studies not performed with sulfamethoxazole and trimethoprim in combination. Trimethoprim demonstrated to be nonmutagenic in the Ames assay. No chromosomal damage observed in human leukocytes *in vitro* with sulfamethoxazole and trimethoprim alone or in combination; concentrations used exceeded blood levels of these compounds following therapy with Bactrim. Observations of leukocytes obtained from patients treated with Bactrim revealed no chromosomal abnormalities. *Impairment of Fertility:* No adverse effects on fertility or general reproductive performance observed in rats given oral dosages as high as 70 mg/kg/day trimethoprim plus 350 mg/kg/day sulfamethoxazole.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Trimethoprim and sulfamethoxazole may interfere with folic acid metabolism; use during pregnancy only if potential benefit justifies potential risk to fetus. Nonteratogenic Effects: See CONTRAINDICATIONS section.

Nursing Mothers: See CONTRAINDICATIONS section.

Pediatric Use: Not recommended for infants under two months (see INDICATIONS and CONTRAINDICATIONS sections).

ADVERSE REACTIONS: Most common are gastrointestinal disturbances (nausea, vomiting, anorexia) and allergic skin reactions (such as rash and urticaria). **FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS (SEE WARNINGS SECTION).** *Hematologic:* Agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, neutropenia, hemolytic anemia, megaloblastic anemia, hypoprothrombinemia, methemoglobinemia, eosinophilia. *Allergic Reactions:* Stevens-Johnson syndrome, toxic epidermal necrolysis, anaphylaxis, allergic myocarditis, erythema multiforme, exfoliative dermatitis, angioedema, drug fever, chills, Henoch-Schoenlein purpura, serum sickness-like syndrome, generalized allergic reactions, generalized skin eruptions, photosensitivity, conjunctival and scleral injection, pruritus, urticaria and rash. Periarteritis nodosa and systemic lupus erythematosus have been reported. *Gastrointestinal:* Hepatitis (including cholestatic jaundice and hepatic necrosis), elevation of serum transaminase and bilirubin, pseudomembranous enterocolitis, pancreatitis, stomatitis, glossitis, nausea, emesis, abdominal pain, diarrhea, anorexia. *Genitourinary:* Renal failure, interstitial nephritis, BUN and serum creatinine elevation, toxic nephrosis with oliguria and anuria, crystalluria. *Neurologic:* Aseptic meningitis, convulsions, peripheral neuritis, ataxia, vertigo, tinnitus, headache. *Psychiatric:* Hallucinations, depression, apathy, nervousness. *Endocrine:* Sulfonamides bear certain chemical similarities to some goitrogens, diuretics (acetazolamide and the thiazides) and oral hypoglycemic agents; cross-sensitivity may exist. Diuresis and hypoglycemia have occurred rarely in patients receiving sulfonamides. *Musculoskeletal:* Arthralgia, myalgia. *Miscellaneous:* Weakness, fatigue, insomnia.

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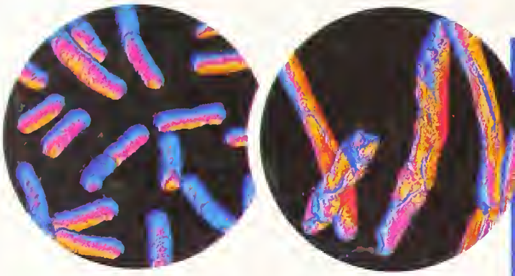

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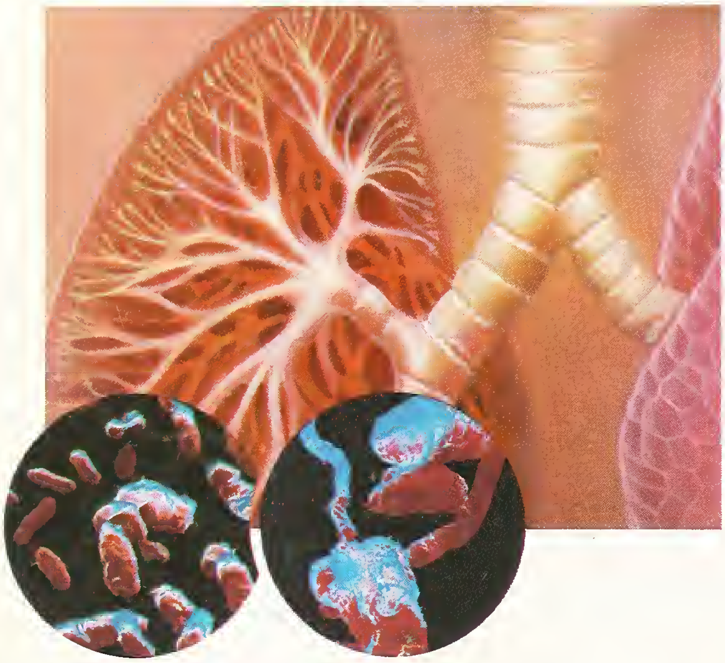

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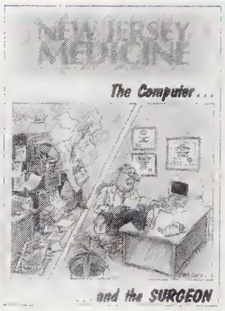

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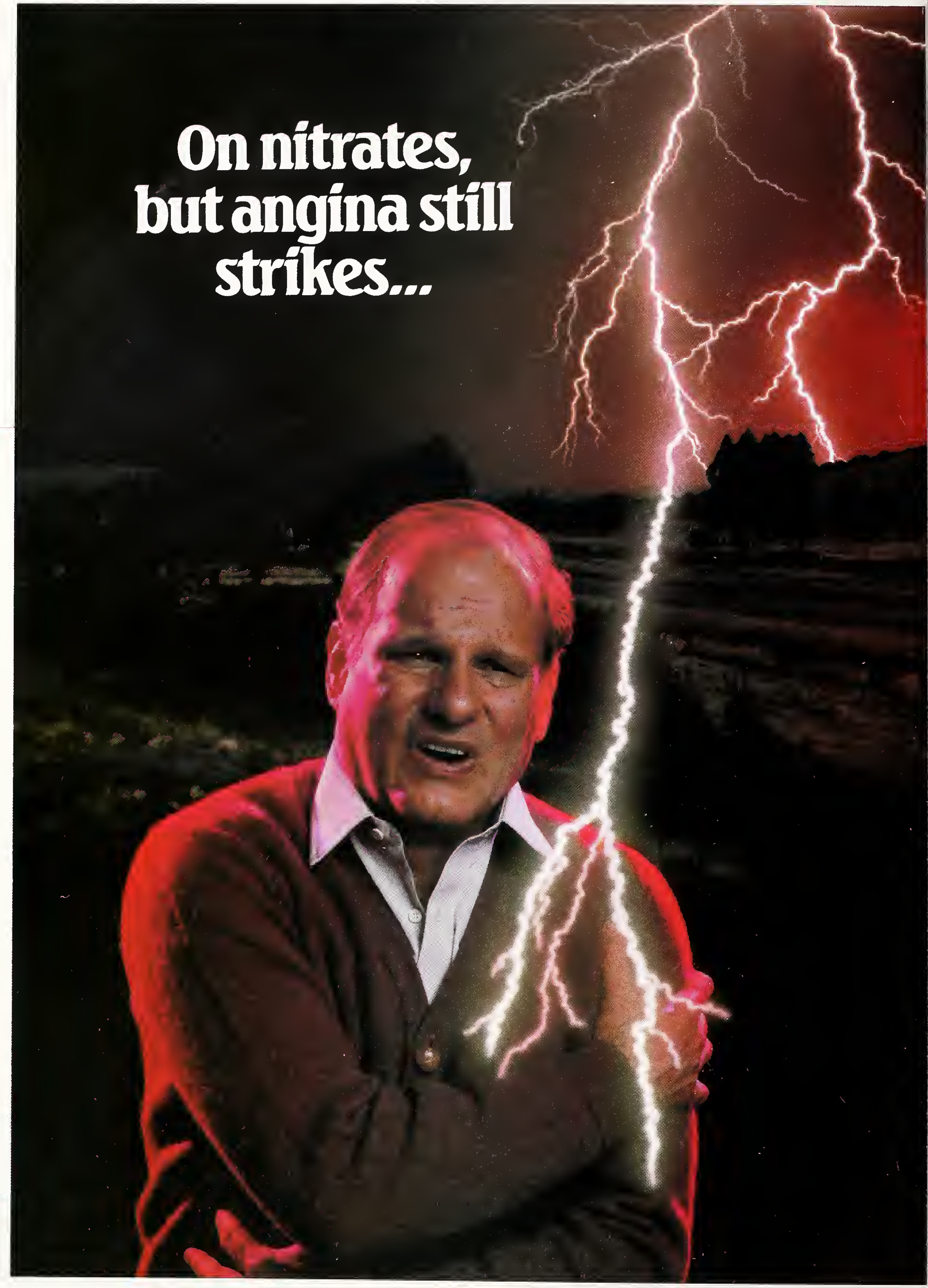
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Marked 1° or progressive 2° or 3° AV block requires a dosage reduction or, rarely, discontinuation and institution of appropriate therapy depending upon the clinical situation. Patients with hypertrophic cardiomyopathy (IHSS) received verapamil in doses up to 720 mg/day. It must be appreciated that this group of patients had a serious disease with a high mortality rate and that most were refractory or intolerant to propranolol. A variety of serious adverse effects were seen in this group of patients including sinus bradycardia, 2° AV block, sinus arrest, pulmonary edema and/or severe hypotension. 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One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. *Pregnancy Category C:* There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use. **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia: HR < 50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%), elevations of liver enzymes have been reported (See *Warnings.*) The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: ecchymosis, bruising, gynecomastia, psychotic symptoms, confusion, paresthesia, insomnia, somnolence, equilibrium disorder, blurred vision, syncope, muscle cramp, shakiness, claudication, hair loss, macules, spotty menstruation. **How Supplied:** ISOPTIN (verapamil HCl) is supplied in round, scored, film-coated tablets containing either 80 mg or 120 mg of verapamil hydrochloride and embossed with "ISOPTIN 80" or "ISOPTIN 120" on one side and with "KNOLL" on the reverse side. 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PROFESSIONAL LIABILITY COMMENTARY*

Blood Transfusions and Patients' Rights

Jehovah's Witness Who Refused Transfusion; Transfusion for Mother and Baby; Adult Patient's Right To Refuse Transfusion; Refusal to Consent

HUSBAND OF JEHOVAH'S WITNESS WHO REFUSED BLOOD TRANSFUSION SUES PHYSICIAN

A physician was not released from liability for negligence by a patient's refusal to receive a blood transfusion, the Washington Supreme Court ruled.

The physician recommended that the patient have a dilatation and curettage (D&C) after diagnosing a missed abortion. The physician did not discuss alternative methods of performing the procedure, but advised the patient that she might bleed during the procedure. The patient sought a second opinion and was advised that the D&C was the appropriate treatment. The second physician also advised the patient of the possibility of bleeding.

The patient and her husband, who were Jehovah's Witnesses, signed a form refusing to permit blood transfusions and releasing the hospital and physician from responsibility for unfavorable results due to this refusal. During the operation, the physician severely lacerated the patient's uterus. The patient began to bleed profusely. She was coherent at the time, and she and her husband both refused permission to give her a transfusion. She bled to death.

The patient's husband brought a wrongful death action alleging that the physician's negligence proximately caused the patient's death. The jury found the physician negligent and that his negligence was a proximate cause of the patient's death. However, the jury found that the patient and her husband knowingly assumed the risk of bleeding to death and attributed 75 percent of the fault for her death to the refusal to permit blood transfusions, reducing the damages awarded from $412,000 to $103,000.

On appeal, the court found the refusal to permit transfusions to be valid and that the document was signed voluntarily. However, the court disagreed with the physician's assertion that the refusal acted as a complete bar to the wrongful death claim. The court pointed out that while the patient and her husband accepted the consequences resulting from a refusal to have a blood transfusion, they did not accept the consequences of the physician's negligence. The court affirmed the lower court's judgment. 695P.2d116 (Wash. Sup. Ct., Jan. 11, 1985). (*The Citation*, Vol. 51, No. 11, September 15, 1985)

BLOOD TRANSFUSIONS GIVEN TO MOTHER AND BABY DESPITE OBJECTIONS

A hospital and attending physicians were entitled to give blood transfusions to a baby and mother during a cesarean section despite religious objections, a New York trial court ruled.

The mother's pregnancy was complicated by anemia, an Rh-negative blood type, and the anterior position of the placenta. The cesarean section delivery was to be premature because of mounting pressure on the baby's brain caused by hydrocephalus. The mother and father wanted all necessary surgical procedures to be carried out with the exception of blood transfusions. The hospital petitioned for an order authorizing necessary blood transfusions to both mother and child The attending physician testified that the cesarean section might entail the loss of a life-threatening amount of blood by the mother.

The court said that the highest state court had made it clear that the state had a vital interest in the welfare of children that would override even the parents' most fervently held religious beliefs. Authorizing the hospital and physicians to give blood transfusions to the baby as medically indicated, the court said that the courts could not permit a parent to deny a child treatment for a life-threatening condition.

As to the mother, the court said that she wanted the hospital and physicians to take aggressive medical steps to insure a proper delivery but did not want them to correct a grave condition that might be encountered in the process. The court said that this put the hospital and physicians in an untenable position. The court held that the attending physicians could continue to administer blood transfusions to the mother even after the moment of delivery as necessary to stabilize her condition. The court said that the mother's freedom to direct the course of her own treatment would be interdicted only in the postoperative period and only for as long as was medically indicated to stabilize her condition. 485 N.Y.S.2d 443 (N.Y. Sup. Ct., Jan. 15, 1985). (*The Citation*, Vol. 51, No. 11, September 15, 1985)

PATIENT HAS RIGHT TO REFUSE TRANSFUSION

An adult patient had a right to refuse a blood transfusion without which he might die within a few hours, a Florida appellate court ruled.

The patient, who was 27 years old, underwent regular renal dialysis for kidney disease. He was obligated to pay $50 per week in support of his only minor child, who lived with his former wife in another state. He was a Jehovah's Witness and believed that ingestion of whole blood would deny him resurrection and eternal salvation.

---

*This item from the Department of Professional Liability Control, MSNJ, was prepared by James E. George, M.D., J.D., and A. Ronald Rouse, who are, respectively, Director of the Department and Director of Special Projects.

The state, the treating physician, and the hospital were apprehensive as to the legal consequences of their acquiescence in the patient's refusal of treatment and concerned for his welfare. They brought an action to determine the patient's right to refuse a transfusion, and the trial court upheld the patient's right to refuse.

On appeal, the appellate court said that, under the facts presented, the patient, who was competent, had the right to refuse a transfusion regardless of whether his refusal arose from fear of an adverse reaction, religious belief, recalcitrance, or cost. The court said that his refusal of treatment could not be categorized as abandonment of his minor child because he seldom saw her and there was evidence that the mother and both families would help support her.

Further, the patient had a small annuity that named the child as beneficiary. The court said that his refusal could not be classed as attempted suicide because of his willing acceptance of dialysis treatment, without which he would die. Affirming the trial court's judgment, the court said that those who practiced, dispensed, or furnished medical services should not be held criminally responsible or civilly liable because they acceded to the patient's refusal to undergo treatment. 465 So.2d 666 (Fla. Dist. Ct. of App., March 27, 1985). (*The Citation*, Vol. 51, No. 11, September 15, 1985)

NEW JERSEY AND REFUSAL TO CONSENT

Physicians treating critically ill or injured patients, who based on their religious beliefs refuse to consent to blood transfusions, are confronted with a myriad of complex problems.

Does an adult patient have a legal right to choose death as a consequence of adhering to religious tenets? May the courts force an adult patient to submit to lifesaving blood transfusions over religious convictions? May parents, in adherence to their religious beliefs, refuse to consent to necessary blood transfusions for their minor children?

In general, a competent adult is free to reject lifesaving medical treatment. This freedom is based on the constitutionally protected right to determine what shall be done with one's body. Freedom to choose, however, may be limited by an overriding or "compelling" state interest.

Courts also have held that the constitutionally protected right to religious belief is absolute, but that one's right to practice one's religion may be limited where necessary to protect the rights of others (98 U.S. 145 (1878).

In the past 25 years, New Jersey courts have decided a number of cases involving the refusal of Jehovah's Witnesses to submit to blood transfusions for themselves or their children.

In 1962, parents who were Jehovah's Witnesses consented to surgery to correct their infant's ventriculoseptal defect. They refused, however, to consent to blood transfusions. Treating physicians believed that without a transfusion the child had only an "outside chance for surviving" and if he did survive, his mental functioning would be impaired.

The hospital applied to the court for appointment of a special guardian for the child. Subsequently, a court ordered the transfusion. Despite the administration of the transfusion, the child died.

The parents appealed, arguing among other things, that their constitutional rights of religious expression had been violated. The Supreme Court of New Jersey disagreed, holding that "the right to practice religion freely does not include liberty to expose the child to ill health or death." It ruled, "That parents may not, in the name of religion, make martyrs of their children before they have reached the age of full and legal discretion when they can make that choice for themselves (37 N.J. 463)."

In 1964, the New Jersey Supreme Court approved an involuntary transfusion where a hospital produced credible evidence that a pregnant patient, a Jehovah's Witness, would probably die if no transfusions of whole blood were administered (42 N.J. 421).

In a 1971 case, the New Jersey Supreme Court upheld a lower court which ordered a blood transfusion for a 27-year-old Jehovah's Witness who was the victim of an auto accident. The patient was disoriented due to her injuries and was unable to assert her wishes. Her mother refused to consent to the transfusions. A lower court appointed a guardian who consented to the transfusion. The Supreme Court of New Jersey found that the state had a compelling and overriding interest in preserving the patient's life. It held that the state's interest and the interest of the hospital staff outweighed the patient's right to assert her convictions (58 N.J. 576). In 1985, the New Jersey Supreme Court expressly recognized the right of a terminally ill adult to reject medical treatment and overruled its decision in the 1971 case to the extent that it implied the contrary (matter of Conroy, 98 N.J. 321 at 351).

In 1974, a lower court in New Jersey ordered that physicians could transfuse a premature infant and accomplish a complete blood exchange over the refusal of his parents who were Jehovah's Witnesses. The court based its determination on medical evidence that to a reasonable degree of medical certainty, the withholding of a complete blood exchange would have led to irreparable brain damage and severe mental retardation. The court observed that had the disputed procedure involved a significant risk of damage to the infant, due to the procedure itself, the parent's religious convictions would have been respected (128 N.J. Sup. 498 Law Div).

In summary, the courts of New Jersey are likely to uphold the right of mentally competent adult Jehovah's Witnesses to refuse lifesaving blood transfusions. Much more problematic are cases involving minor children of Jehovah's Witnesses and adults whose condition renders them unable to express their wishes. We may expect courts to continue to appoint guardians for the minor child and to hold bedside hearings where necessary to ascertain the necessity of the proposed medical procedures where such have been refused by parents based on their own religious beliefs. Applications to the court for emergency medical relief usually are handled by the hospital's attorney who may seek a court order for emergency treatment of minors or incompetent adults.

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This Is Your Year—MSNJ 1986 Annual Meeting

The 1986 Annual Meeting of the Medical Society of New Jersey will be held May 1 to May 4 at Host Farm Resort.

For reasons beyond the control of the Committee on Annual Meeting and the staff, a move of location was obviously necessary because the Bellevue Stratford Hotel closed. A number of alternatives, including other Philadelphia locations, Atlantic City, Glen Pointe, Cherry Hill, and Host Farm, were considered carefully. After extensive negotiations, all were eliminated as a satisfactory site except one. The Executive Committee of the Board of Trustees approved the Host Farm Resort on May 1 to May 4, 1986, as the location and dates for the 1986 Annual Meeting of the Medical Society of New Jersey.

If you have never attended an Annual Meeting, this is your year! Like the fundraisers for great symphony orchestras are likely to warn their constituents—the music might stop. Members of the Medical Society who go their merry way and do nothing to help themselves, their profession, or their colleagues are likely to be facing a wall of molten bureaucratic lava with two bare hands and nobody to help.

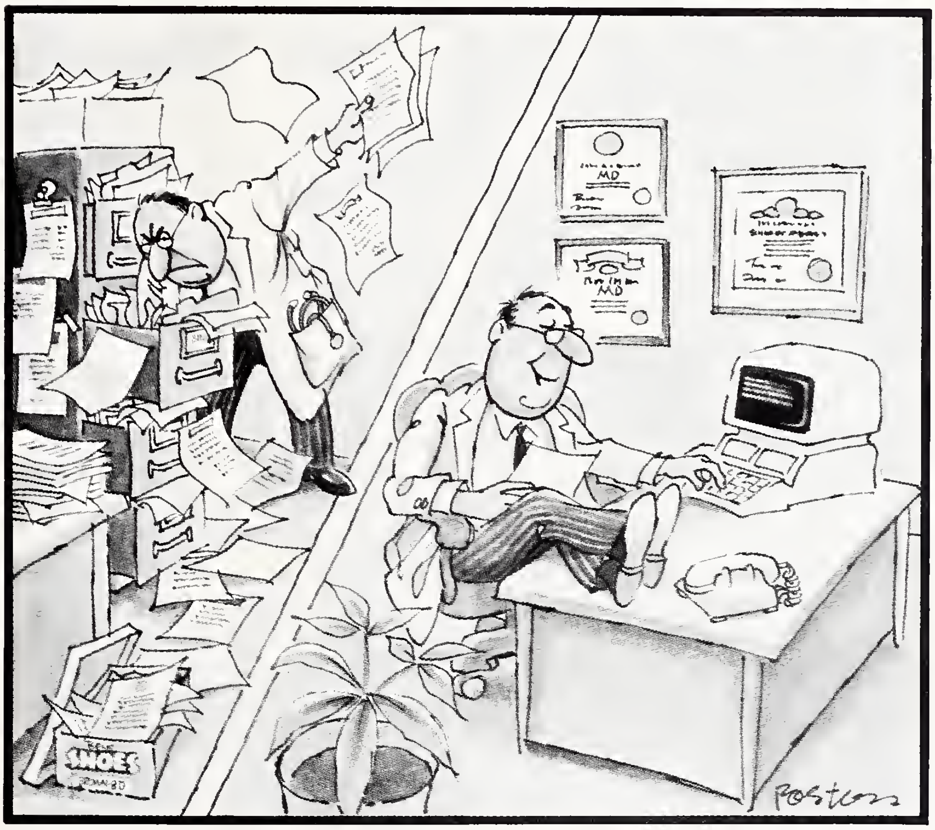
The pitiful query of the inactivist—What has the AMA ever done for me?—is the rhetorical question of a desperate individual who perceives an irremediable situation. What shall he do? Quit medicine? Join an HMO? Get a job? Form a trade union of physicians? Discourage his children from becoming physicians? If you have had these thoughts and feelings, come to the Annual Meeting!

Are you not aware of the major social events of the past four decades that have altered the relationships of the medical profession to society at large? Do you not recognize that the autocratic physician of eastern European tradition no longer is acceptable to the American public? Do you resent the loyal patient who seems to doubt you by timidly requesting a second opinon? Do you cringe at the pressures of government intervention, union demands for first dollar coverage (which is largely responsible for the public attitude of today), and the thought of negligence claims and malpractice insurance rates?

Think about what you have done for the medical profession in the past 10, 20, 30, or 40 years. Have you been giving or taking, without ever expressing a "thank you" to your peers who have attended monthly board meetings and spent thousands of hours of travel and committee meeting time at a cost to their practices, their income, and their recreation and family time? Have you expected your leaders to move mountains in your behalf without raising a finger to point to the mountain you want moved?

Ladies and gentlemen of New Jersey medicine—this is the year! Come to Host Farm Resort and change from an inactivist to an activist. A.K.

THE COMPUTER and the SURGEON



In 1981, I described nonbusiness-related uses of a popular microcomputer in "TRS-80 and the Surgeon."[1] Since then the electronic revolution has progressed rapidly and proliferation of hardware and software makes an update now appropriate.

The mammoth IBM Corporation stepped into the more affordable computer range with the PC, XT, PC Jr., and AT models. Currently, I use the PC with 256 kilobyte memory capacity, two 5¼-inch floppy disk drives, an IBM color monitor, and an Epson FX-80 matrix printer. This complete system can be purchased for around $4,000 and has proved highly reliable. The "do-it-yourselfer" can add peripheral devices; however, it is relatively easy to disconnect the cables and bring the computer components to local retail outlets for service. For purchasing new hardware or upgrading the system, one can shop the newspapers or the magazines which advertise mail-order discounted hardware and software programs.

DONALD ROTHMAN M.D.

Desktop computer programs that store and process medical information and allow rapid access to large current data bases represent exciting developments for the harried clinician attempting to keep up with a practice.

The main use of the desktop computer in my office remains data storage. Recently, I went to the Lotus 1-2-3 (Lotus Development Corp, Cambridge, MA) integrated software program and its straightforward row-column format has proved easy to learn for the basic matter of data entry. I prefer this to the earlier Visifile program for its ease of use, less manipulation of disks, and overall rapid performance. These aspects are enhanced by the added capabilities of graphing, calculating, and basic statistical analysis. For writing scientific papers of a clinical nature this would seem an ideal tool.[2] The spreadsheet portion is for basic accounting manipulations. I continue to monitor my surgical cases with emphasis on colon and breast cancer. Separate files can be made up for more concentrated study of any single disease state.

An inexpensive program called PC-Crayon allows production of colorful screen formats—pictorial and printed material—which can be photographed for slide talks. There are specifically designed cameras and masking devices to photograph computer screens. Printouts in black and white can be photographed as well. Running the matrix printouts on your copier will darken the blacks for better reproduction. There is a signmaker (Sign Designer, Power-Up, Half Moon Bay, CA) that will make posters in three print types that can be copied (transferred) onto transparencies by your copy machine for overhead projectors. These can be made up in a short time for lectures by avoiding the middleman. I photograph the screens with a Nikon FE2 and zoom lens using a plastic bag to block out reflected room highlights. One can, for example, make slides of white on blue or black with striking visual effect for 33 mm projections that are nearly professional quality with less expense and time. Polaroid makes an instant 35 mm slide film and mounting system for on the spur-of-the moment productions.

I use Volkswriter (Lifetree Software Inc., Monterey, CA)—a word-processing program—to write my scientific papers and medical expositions.

If one has not tried computer typing with a word processor, one is missing the delight of rapid, quiet composition, ease of correction, and flexibility. Storage of the data for later rewriting and then printing makes computer use a strong competitor of typing, handwriting, and dictation methods. Consultation letters and any repetitive printed material can be set up on the computer. Mailing lists and address labels can be stored and utilized this way. Elimination of paper with compact electronic storage is a plus and some offices already use computers for history taking and basic followup. Newer generations of physicians will find computer filing more acceptable than the doctors who grew up on paper and cumbersome filing cabinets. Loss of electronic data can be eliminated by back-up procedures and hard copy printouts of important data. It is easier to store compact electronic data in fireproof containers (than heavy paper material) and take disks to another location.

An internal computer board allows me to use a Hayes (Hayes Microcomputer Products Inc., Norcross, GA) modem 300 baud with addition of a phone company-installed connection to access data over phone lines. A nearly free system of data retrieval is offered by Phycom Service (Fisher-Stevens, Inc., Totowa, NJ). I presently subscribe to BRS-Saunders Colleague which allows tapping into Medline and other health-related data libraries. In addition, I can tie into PDQ which has an update of current cancer treatments. One must familiarize oneself with the software and learn how to do medical searching. The effort is worthwhile since it channels one's thinking and lets you know what is out there. Through BRS one can electronically access medical journals and texts. One can work up a bibliography and find experts in areas of interest. This would be useful to professionals who are not near a good library or librarian. Availability of this source is a true advantage when information is needed on short notice for complex or unusual problems. Hard copies of the accessed data can be made on the spot on your printer or received from BRS through the mail. Phone charges to local numbers on Telenet and Uninet are reasonable.

SUMMARY

The electronic age is upon us. Many disciplines are extensive computer users. Physicians should join the electronic era. Your personal computer and the world of information and communication are at your fingertips.

REFERENCES

1. Rothman D: TRS-80 (TM) and the surgeon. *J Med Soc NJ* 78: 538, 1981.
2. Sladen JG: The personal computer as a clinical research and teaching tool. *Am J Surg* 147:654-659.

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CARDIZEM® 60 mg tid or qid

diltiazem HCl/Marion

FEWER SIDE EFFECTS IN ANTIANGINAL THERAPY

BRIEF SUMMARY

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist).

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM (diltiazem hydrochloride) in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted.

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy** — CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

CARDIZEM 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other. Issued 4/1/84

See complete Professional Use Information before prescribing

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- ***Proven efficacy when used alone in angina[1, 4-6]***
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Please see brief summary of prescribing information on the next pa

DOES THEOPHYLLINE IMPROVE DYSPNEA IN NONREVERSIBLE COPD?

BURTON M. COHEN, M.D., ELIZABETH*

Sustained-release oral theophylline may improve breathlessness in a subset of patients with chronic nonreversible lung disease and elevated nasal airways flow/resistance by direct nasal effects.

Theophylline therapy may improve symptoms and lessen dyspnea in nonreversible chronic obstructive airway disease with little change in lung function, gas exchange, or exercise performance. Effects on respiratory and ventricular muscles or ventilatory drive have been incriminated.[1-5] Because nasal airflow resistance (R_n) is commonly abnormal in such patients, adding to the respiratory load from the primary lower tract disorder,[6,7] we examined theophylline effects on the elevated R_n of patients with, or without, chronic obstructive lung diseases.

MATERIALS AND METHODS

Nine adults, seven men and two women (mean age 48.0 ± 16.1 years), entered the first trial. They fulfilled the following criteria: 1) perennial allergic rhinitis producing nasal obstruction and mouth breathing for at least two years; 2) allergy confirmed by skin prick tests; 3) no history of bronchial asthma; 4) normal spirometry; and 5) no systemic or topical medication during the previous two months. Individuals with anatomic nasal obstruction were excluded. Nasal airway resistance was determined by posterior electronic rhinometry using a commercial apparatus, and was computed as the mean of triplicate determinations of the sum of inspiratory and expiratory resistances read at the reference flow rate of 0.5 l/sec from the digital displays of the device.† The three test days for each patient were separated by 48-hour washout periods. Baseline measurements were made at 6 A.M. and repeated 1, 3, 5, 7, 9, 10, and 12 hours later either without treatment or following single 200 mg or 400 mg tablet doses of a sustained-release theophylline,‡ according to individual randomized coded assignments.

The second trial included six patients, five men and one woman (mean age 68.0 ± 9.4 years), with nonallergic chronic obstructive lung diseases[9] receiving long-term steady-state rations of 800 mg of the same theophylline preparation as a single morning dose. These individuals demonstrated less than a 15 percent rise in $FEV_{1.0}$ after two inhalations of a standard isoproterenol aerosol[10] and R_n values exceeding 3.00 cm H_2O/1/sec on at least two separate measurements of each index during the week before entry. These patients were studied once, before, and 1, 3, 5, 7, 9, 10, and 12 hours after their usual morning theophylline. Their measurements by electronic posterior rhinometry were made with our own apparatus[11] substituting a Scott No. 801450-13 Aviation Mask* modified for equipment connections for the original oronasal mask.[12] The traces inscribed on the precalibrated axes of the "memory-type" oscilloscope were photographed (Figure 1). Venous blood was drawn 10 hours after dosing for measurement of serum theophylline concentration.** Aerosolized steroids or oral or inhalational β-adrenergic agents were forbidden 12 hours before, and during, the trial; patients on chronic oral steroids were excluded. Smoking was not allowed for both groups, and only water was permitted during the first 4 hours of study; a light lunch, omitting xanthine-containing beverages, was scheduled 5½ hours after baseline to allow a 1-hour period of reacclimatization before the hour 7 determination.

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*This article was presented at the 13th Annual Meeting of the New Jersey Thoracic Society, June 5, 1985. Dr. Cohen is Clinical Professor of Medicine, UMDNJ-New Jersey Medical School, and Senior Attending, Elizabeth General Medical Center. Correspondence may be addressed to Dr. Cohen, 425 Westfield Avenue, Elizabeth, NJ 07208.

†Quintron Nasal Airway Resistance Computer (NARC), Quintron Instrument Company, 3712 W. Pierce St., Milwaukee, WI 53215.

‡The 200 and 400 mg tablets of Uniphyl®, The Purdue Frederick Company, Norwalk, CT 06856, were purchased locally.

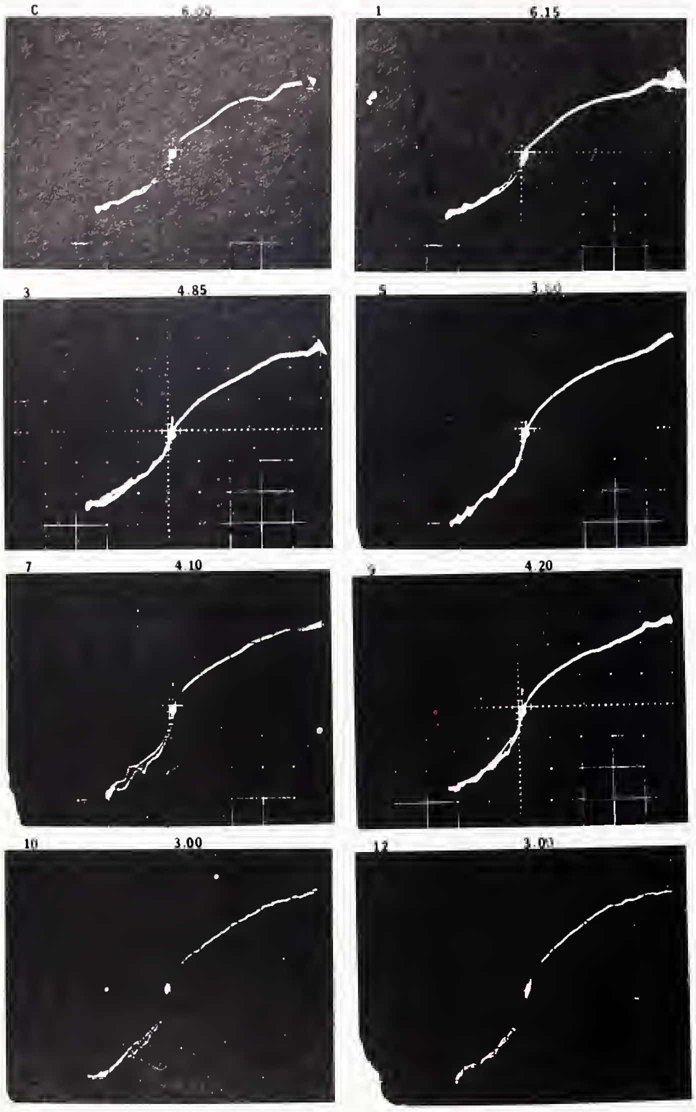

Figure 1—Oscilloscope camera photographs recording nasal flow resistance traces of a 79-year-old man with chronic bronchitis at baseline and at 1, 3, 5, 7, 9, 10, and 12 hours following his steady-state morning dose of 800 mg of a sustained-release theophylline preparation. Time is given in left upper corner of each photograph and nasal flow resistance (in cm $H_2O/1/sec$) above the middle of the trace.

The student's t-test was used to evaluate differences between baseline and post-dosing values—to compare the baselines for the rhinitis protocol and possible differences between untreated and active drug responses at each time period.

RESULTS

Nasal resistance behavior in the rhinitis trials. All nine patients completed their individual protocol assignments. Table 1 lists the mean absolute R_n values and their standard deviations serially during each of the three test days and statistically significant changes from baseline at the 95 percent confidence level or better. The 6 A.M. means of 5.76, 5.92, and 6.03 cm $H_2O/1/sec$ did not differ. Without treatment, R_n rose steadily, reaching a mean increase of 12.2 percent above baseline 12 hours later. On the theophylline treatment days, R_n fell, with the maximum decrement for both doses 9 to 12 hours from baseline (Figure 2). While only the means after the 400 mg dose could be differentiated statistically from baseline, both active treatments induced R_n responses significantly different from the untreated means (P=0.05 or better) from 3 to 12 hours for 400 mg, and 5 to 12 hours for the 200 mg observations.

Nasal resistance responses for COPD patients. For the patients with chronic lung disease, R_n fell from the control mean of 5.51 (±0.65) to 3.59 (±1.79) cm $H_2O/1/sec$, a mean decline of 34.8 percent. The changes were different statistically from baseline (P=0.05 or better) from hours 5 to 12 inclusive (Table 2 and Figure 3). The mean serum theophylline concentration at the time of maximum R_n improvement was 15.52 ± 1.56 mcg/ml. Although group mean nasal resistance values were significantly better than baseline over the last 7 hours of observation, R_n rose for one of the six patients over the entire 12 hours, despite an acceptable 10-hour theophylline level of 13.9 mcg/ml.

DISCUSSION

The higher nasal airflow resistance of patients with nonasthmatic expiratory airflow disorders, compared with matched normal individuals[6,7,12,13] (Table 3) originally was thought to be compensatory, i.e. acting in the fashion of "pursed lips" breathing to forestall bronchiolar collapse.[14,15] Measures reducing the abnormal R_n have been considered helpful and safe; appraisal of the behavior of nasal and lower respiratory tract resistances, measured in concert, have been more informative than reliance upon lower airway resistance-based indices alone in defining the site and total activity of respiratory medicines and the relative potency of inhalants.[7,12] Like asthmatics, patients with chronic bronchitis and emphysema developed better nasal patency after exercise, the R_n decrease modifying the rise in lower airway resistance R_a that accompanies exertion; instillation of decongestant nose drops before exercise increased the response in the nose and further damped the postexertional R_a rise.[13] The speed, magnitude, and direction of these responses was consistent with the operation of a nasal-pulmonary reflex arc.[16,17]

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*Scott Aviation Company, Lancaster, NY 14806, obtained from Guardian Safety Equipment Company, Linden, NJ.

**Serum theophylline concentrations for venous blood were determined by the Therapeutic Drug Monitoring System (TDx, Theophylline Reagent System, Abbott Laboratories) by Metpath Laboratories, Teterboro and Hackensack, NJ.

TABLE 1

Mean Nasal Resistance (R_n) Values and Standard Deviations (in parenthesis) in a Comparison of 2 Sustained-Release Theophylline Doses with No Treatment for 9 Patients with Allergic Rhinitis

		Mean R_n (cm $H_2O/1/sec$) Hours after Dosing						
Treatment	**Control**	**1**	**3**	**5**	**7**	**9**	**10**	**12**
None	5.76 (1.02)	5.67 (0.91)	5.77 (0.86)	5.96 (0.89)	6.07 (0.93)	6.23 (0.89)	6.38 (0.80)	6.46 (0.87)
200 mg	5.92 (0.99)	5.83 (0.91)	5.60 (0.98)	5.44 (1.06)	5.46 (1.10)	5.43 (1.20)	5.21 (1.26)	5.20 (1.33)
400 mg	6.03 (0.88)	5.91 (0.88)	4.90† (1.93)	5.10* (0.90)	4.94† (0.88)	4.76† (0.95)	4.70† (0.95)	4.73† (0.91)

*Statistically different from baseline at the 0.05 level.
†Statistically different from baseline at the 0.01 level or better.

TABLE 2

Mean Nasal Resistance (R_n) Values and Standard Deviations for 6 Patients with Nonreversible COPD before and after their Daily Morning Rations of 800 mg of a Sustained-Release Theophylline

		Mean R_n (cm $H_2O/1/sec$) Hours after Dosing						
Index	**Control**	**1**	**3**	**5**	**7**	**9**	**10**	**12**
Mean	5.51	5.22	4.98	4.13†	4.00†	4.26*	3.59*	4.76*
Standard Deviation	0.65	0.91	0.63	1.11	1.29	0.76	1.79	0.99

*Statistically different from baseline at the 0.05 level.
†Statistically different from baseline at the 0.01 level or better.

TABLE 3

Resistance Values in Normals and Patients with Nonasthmatic Diseases

Investigator	**Diagnosis**	**No. of Pts.**	**R_a***	**R_n***	**$R_a + R_n$***
Nolte & Ulmer[6]	Normal	43	—	3.74	—
(1966)	Bronchitis	29	—	4.72	—
Cohen[7]	Normal	25	1.49	2.47	3.96
(1969)	COPD†	25	3.06	3.99	7.05
Cohen[12]	Normal	50	1.56	2.69	4.25
(1970)	COPD†	50	7.37	4.62	11.99
Cohen[13]	Normal	200	1.52	2.58	4.10
(1971)	COPD†	200	5.97	4.38	10.35

*R_a represents lower airway resistance, R_n represents nasal airways resistance, and $R_a + R_n$ represents their sum, all given in cm $H_2O/1/sec$.
†COPD represents patients with nonasthmatic chronic obstructive airways diseases.

Recently reported effects on mediator release in patients with pollen-induced allergic rhinitis implying local theophylline activity in the nose were not accompanied by changes in R_n determined by anterior rhinomanometry.[18] We have used the posterior technique which is reliable, reproducible, and free of the serious errors of the anterior approach.[19] It agrees with body plethysmography.[20] Our data indicate that this method can sense the effects of theophylline on nasal patency in such patients, that the changes are dose-related and differ significantly from the behavior of the untreated allergic nose. A larger (800 mg) steady-state dose of sustained-release theophylline induced similar improvement in the nasal airway resistance patterns of our six patients with nonreversible chronic obstructive airway disease, with the ten-hour mean percent fall in R_n greater than that of the 400 mg rhinitis trial. The R_n behavior in the former observations and the ten-hour theophylline concentrations are compatible with the known physiologic/pharmacokinetic profile of the preparation used.[21,22] We have shown that an R_n improvement of the magnitude seen is commonly sensed as clinical relief, approximating the figures for acceptable bronchodilatation and for threshold perception of changes in lower tract respiratory loads.[23,24] The situation resembles responses to an intranasally given corticosteroid which decreased nasal obstruction, nasal airway resistance, cough, asthma severity, and exercise-induced asthma with only marginal improvement in lung mechanics.[25]

In reviewing nasal complications of pulmonary or systemic disease and lower airway complications of nasal processes, Proctor speculated that a primary failure of nasal function over a long period "might lead

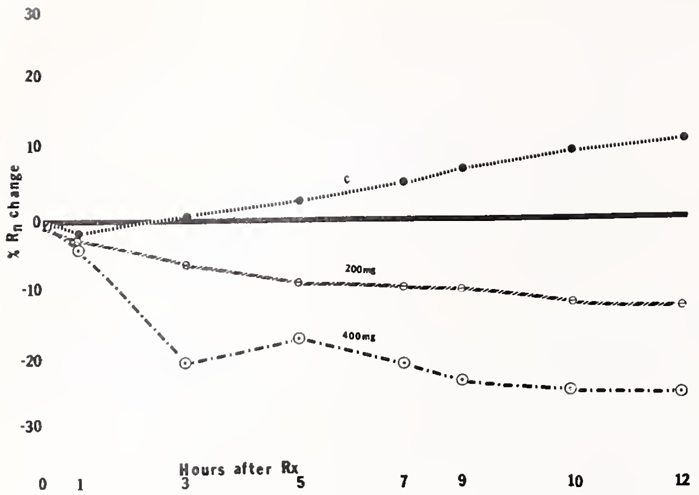

Figure 2—Behavior of nasal airways flow/resistance (R_n) means for nine patients with perennial allergic rhinitis uncomplicated by lower airways disease, untreated (C) and following single tablet doses of 200 and 400 mg of a commercial sustained-release theophylline preparation.

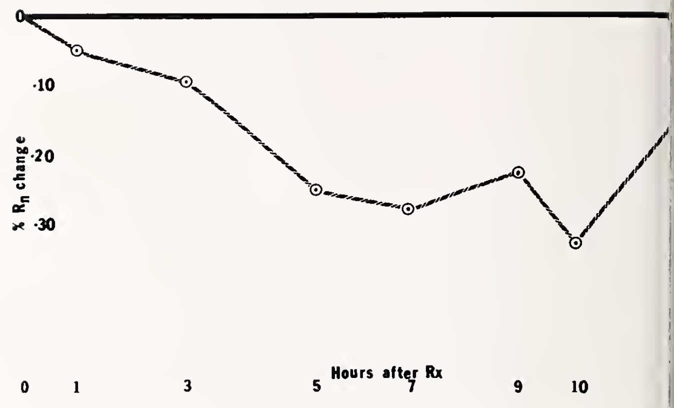

Figure 3—Behavior of mean nasal flow resistance (R_n) for s patients with nonallergic irreversible chronic obstructi lung diseases tested before and after their steady-state mor ing rations of 800 mg of a commercial sustained-relea theophylline preparation.

either to an increased susceptibility to disease or to a gradual deleterious change in the pulmonary airways."[7] In his view, control of nasal airflow resistance, preventing the upper airway malfunction from placing a burden on the compromised lower tract, would appear critical in the management of chronic nonallergic and allergic expiratory airflow diseases. While suggesting the helpful effects of theophylline in reducing the respiratory load, and thereby improving breathlessness, in a subset of patients with chronic nonreversible lung disease and elevated R_n by a direct effect on the abnormal nasal airways dynamics, we do not exclude other physiologic mechanisms, any or all of which may be operative. Our data require confirmation in chronic double-blinded dose-response trials with placebo control and scoring of breathlessness and effort tolerance.

SUMMARY

Theophylline may lessen dyspnea in chronic nonreversible obstructive airway disease with little change in lung tests, gas exchange, or exercise performance. Because nasal airway resistance (R_n) is commonly high in such patients, we examined theophylline effects on patients with R_n elevations by electronic posterior rhinometry. We measured R_n from 6 A.M. to 6 P.M. for nine patients with uncomplicated allergic rhinitis, without treatment and after single doses of 200 and 400 mg of a sustained-release preparation. Six patients with nonallergic expiratory airflow disorders and an insignificant acute $FEV_{1.0}$ response to β-adrenergic aerosol testing were observed over the same period before and after their steady-state daily morning rations of 800 mg of the same drug. The baseline means for the three days of the first trial did not differ; untreated R_n rose 12.2 percent above baseline by 6 P.M. On the 200 and 400 mg days, R_n fell with the maximum decrement for the two doses 8 to 12 hours from control. While only the means following the 400 mg dose differed significantly from baseline, both doses induced R_n deviations significantly different from the untreated means, from 3 to 12 hours for 400 mg and 5 to 12 hours for the 200 mg observation (P=0.05 or better). For the chronic lung patients, F fell from a baseline mean of 5.51 (±0.66) 34.8 percer to 3.59 (±1.79) cm H_2O/1/sec at 10 hours after dosin the mean theophylline concentration at that time wa 15.52 mcg/ml. An improvement in R_n of this magn tude commonly is sensed as clinical relief. These dat suggest that sustained-release oral theophylline ma lessen dyspnea in a subset of patients with chroni nonreversible lung disease and elevated R_n by impro ing the abnormal nasal airways dynamics.

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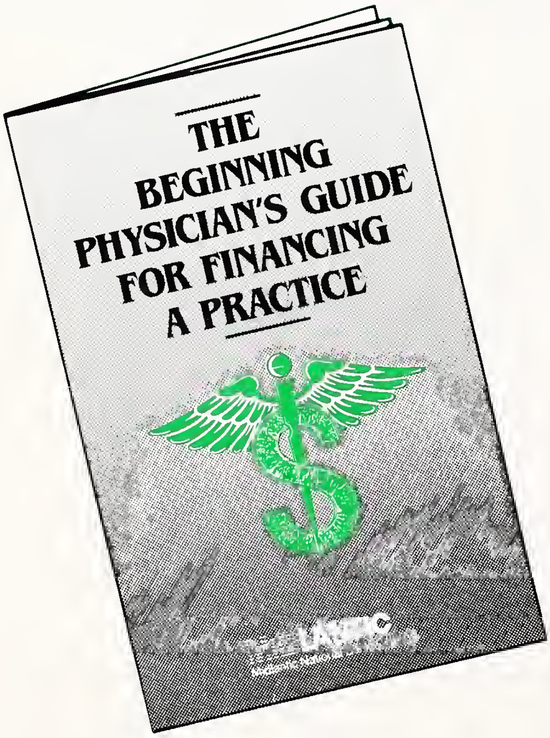

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DIET AND CANCER: ROLE OF LEGUME-DERIVED ANTICARCINOGENS

JONATHAN YAVELOW, PH.D., LAWRENCEVILLE*

The heightened appreciation of the role of diet and cancer among the general population during the last decade has led to an increase in research. The author reviews the epidemiological evidence concerning diet and particularly colon cancer.

There has been a heightened appreciation of the role of diet and cancer among the general population during the last decade. This has led to an increase in research which has culminated in understanding how particular nutrients in the diet may help prevent certain types of tumors. A large literature has evolved pertaining to diet and cancer.[1-3] In this paper I briefly review the epidemiological evidence concerning diet and particularly colon cancer; and trace the brief history concerning the identification of a protein in legumes which possesses an interesting anticarcinogenic potential with respect to colon cancer.

EPIDEMIOLOGY

The correlation between diet and cancer in humans originally was appreciated from epidemiological studies. Japanese immigrants have provided a population group for study in comparing the cancer incidence among Japanese immigrants to America, Japanese living in Japan, and Americans. These studies proved with respect to colon and breast cancer that the Japanese living in Japan had a lower incidence of the tumors and that Japanese living in America assumed the tumor incidence of the United States within a few generations. Individuals used in these studies did not interbreed and, therefore, a genetic effect in the incidence of colon and breast cancer appears not to be the prime risk factor. Presumably there is some factor in the environment which correlates with increased risk.

The classic study of Armstrong and Doll[4] demonstrated a remarkable correlation between meat consumption and the incidence of colon and breast cancer. Their study and others clearly have implicated a high fat diet as a factor increasing the risk for these tumors. Epidemiological studies have been followed up by animal experiments demonstrating a greater incidence of colon and breast tumors as a function of fat intake.[5] The mechanism may involve elevated prolactin in breast carcinogenesis[6] and elevated bile acid levels in colon carcinogenesis.[7] In this regard, epidemiological studies have implicated avenues for experimentation resulting in a more complete understanding of the process of carcinogenesis.

Epidemiological studies also have demonstrated that vegetables and legumes present a negative correlation with respect to colon and breast tumor incidence.[8,9] Is this simply a result of a decreased intake of fat or may these foods contain anticarcinogens? Laboratory experiments have identified anticarcinogens in vegetables as antioxidants[1] and studies suggest anticarcinogens in legumes may be protease inhibitors.[10] Indeed, protease inhibitor-supplemented diets suppress dimethylhydrazine-induced colon tumors in mice.[11]

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LEGUME-DERIVED PROTEASE INHIBITORS

The protease inhibitor common to all legumes is the Bowman-Birk trypsin and chymotrypsin inhibitor.[12] The Bowman-Birk designation is derived from those researchers who discovered the protein. The molecule is present at highest concentrations in soybeans but structurally similar protease inhibitors (referred to as members of the Bowman-Birk family of protein protease inhibitors) are present in chick peas, kidney beans, lentils, and peanuts. This protein is heat stable and, therefore, stable to cooking.[13] In studies of the metabolic fate of immunoreactive ^{125}I—Bowman-Birk soybean inhibitor fed to chickens, it has been reported that 20 percent of the inhibitor was recovered in the intestinal tract and feces.[14] We have extended these studies to rats and mice to determine the distribution of the ingested inhibitor. We have used high-pressure liquid chromatography and affinity chromatography to characterize the labeled Bowman-Birk inhibitor found in the feces and cleaned colon wall.[15,16] It was noted that after feeding the inhibitor, 73 percent of the label accumulates in the intestinal tract and feces. That which is absorbed is not active as a protease inhibitor. The major radioactive inhibitor eluted with a molecular weight of 33,000 daltons. This is the precise elution position shown by trypsin or chymotrypsin complexed with the inhibitor formed in vitro. Moreover, when these complexes were acidified to pH 2.5, a condition which dissociates complexes between protease and protease inhibitor, a 16,000 dalton radioactive material was formed identical to the Bowman-Birk inhibitor, which occurs as a dimer in solution. Thus, this typical protease inhibitor from the soybean survives inactivation by the stomach and appears active in the intestinal tract and colon.

In addition to their stability to cooking and digestion, the Bowman-Birk type protease inhibitors are capable of affecting cells in vitro. We have reported nanomolar (10^{-9} *M*) levels of protease inhibitors suppress x-ray-induced malignant cell transformation in vitro.[17] This suggests a protease is involved in the mechanism of malignant cell transformation. Thus far, we have identified the chymotrypsin-inhibitory site on the Bowman-Birk inhibitor to be involved in the suppression of transformation. As long as four to five days after x-ray treatment the Bowman-Birk inhibitor effectively suppresses malignant transformation. Thus, subsequent to carcinogen exposure the protease inhibitor represents a protein capable of acting as an anticarcinogen. Experiments suggest an extract containing the Bowman-Birk inhibitor is capable of blocking dimethylhydrazine-induced colon tumors in mice.[18]

CONCLUSION

The original epidemiological studies concerning migrating populations are interesting with respect to legumes. Japanese consume less meat and more soy protein (legume) than the typical American, thus, a low fat/protease inhibitor-rich diet may account for the low incidence of colon and breast cancer in Japan as compared to in the United States. However, it is well known that colon and breast cancer run in families. Is this because dietary habits are passed on from generation to generation or might diet modulate one's genetic predisposition toward a certain type of tumor? Recent studies suggest dietary calcium can modify the proliferation of colonic epithelium from high-risk individuals for familial colon cancer.[19] This experiment suggests that one's diet can modulate even genetic predisposition to cancer. More experimentation into the nature of this modulation is necessary.

Epidemiological studies indicate vegetarians to be at low risk for colon and breast cancers. It is tempting to speculate with respect to colon cancer that the Bowman-Birk family of protease inhibitors may have a direct influence in this epidemiological observation.

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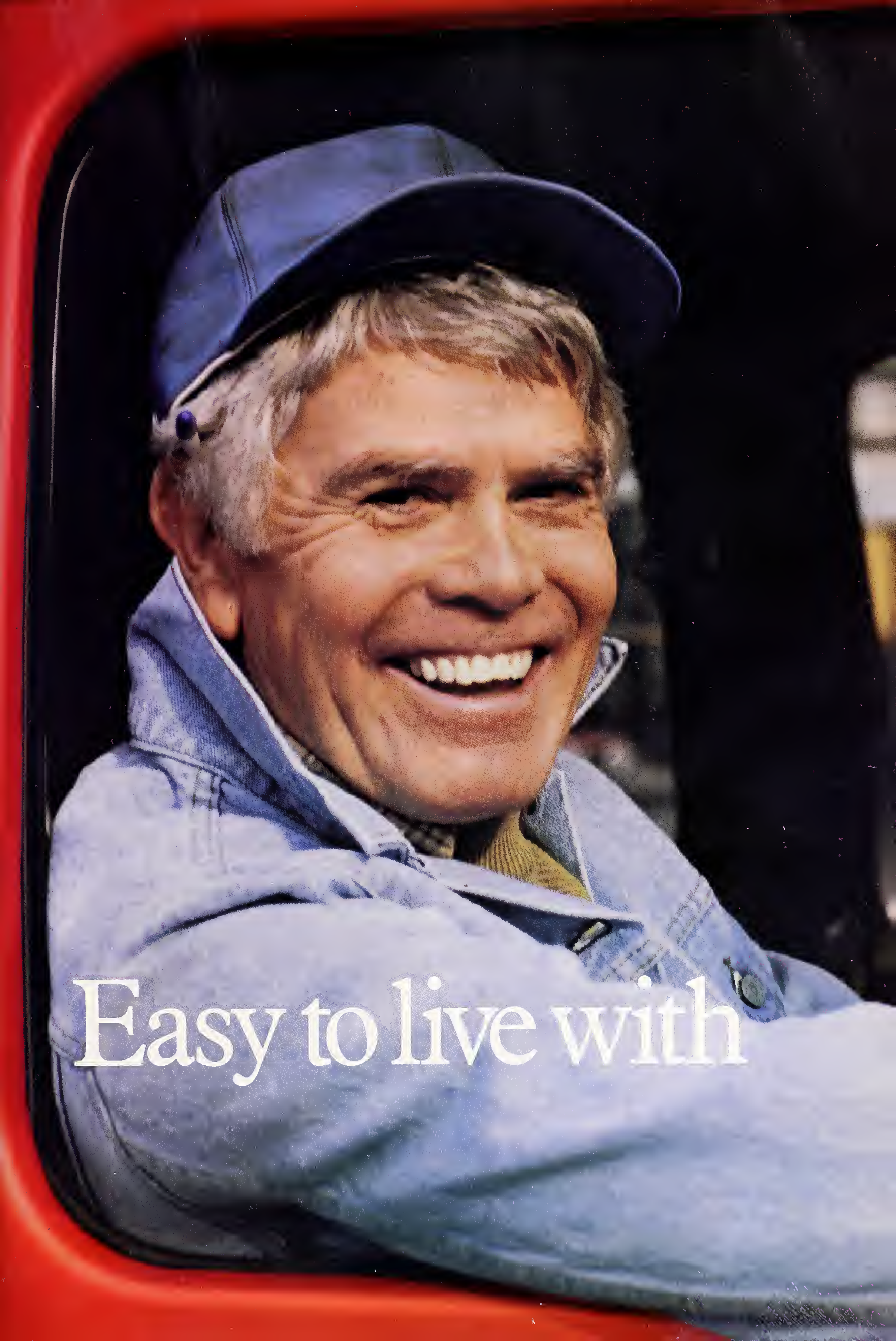
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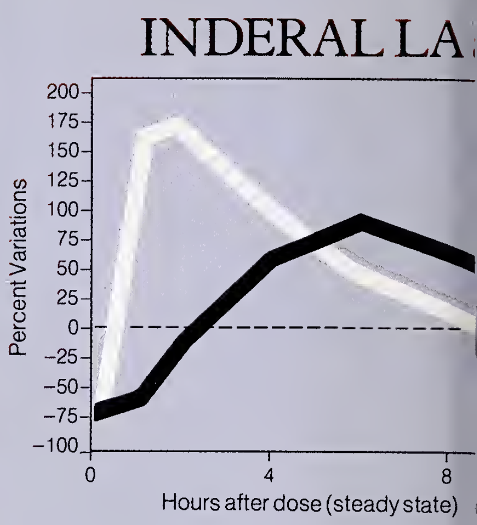

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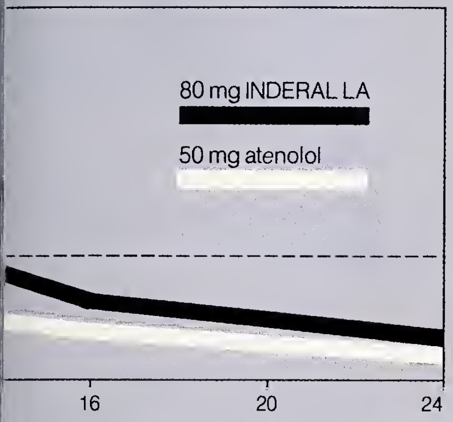

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CONTRAINDICATIONS

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Hydrochlorothiazide: Hydrochlorothiazide is contraindicated in patients with anuria or hypersensitivity to this or other sulfonamide-derived drugs.

WARNINGS

Propranolol hydrochloride (INDERAL® LA): CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated, and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or propranolol should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction following *abrupt* discontinuance of propranolol therapy. Therefore, when discontinuance of propranolol is planned the dosage should be gradually reduced and the patient carefully monitored. In addition, when propranolol is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If propranolol therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute propranolol therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD, IN GENERAL, NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution, since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

DIABETES AND HYPOGLYCEMIA: Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin. Hypoglycemic attacks may be accompanied by a precipitous elevation of blood pressure.

Hydrochlorothiazide: Thiazides should be used with caution in severe renal disease. In patients with renal disease, thiazides may precipitate azotemia. In patients with impaired renal function, cumulative effects of the drug may develop.

Thiazides should also be used with caution in patients with impaired hepatic function or progressive liver disease, since minor alterations of fluid and electrolyte balance may precipitate hepatic coma.

Thiazides may add to or potentiate the action of other antihypertensive drugs. Potentiation occurs with ganglionic or peripheral adrenergic-blocking drugs.

Sensitivity reactions may occur in patients with a history of allergy or bronchial asthma.

The possibility of exacerbation or activation of systemic lupus erythematosus has been reported.

PRECAUTIONS

Propranolol hydrochloride (INDERAL® LA): GENERAL: Propranolol should be used with caution in patients with impaired hepatic or renal function. Propranolol is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that propranolol may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs, such as reserpine should be closely observed if propranolol is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity, which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies, in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. Propranolol has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximal recommended human dose. There are no adequate and well-controlled studies in pregnant women. Propranolol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

NURSING MOTHERS: Propranolol is excreted in human milk. Caution should be exercise propranolol is administered to a nursing mother.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

Hydrochlorothiazide: GENERAL: Periodic determination of serum electrolytes tc possible electrolyte imbalance should be performed at appropriate intervals.

All patients receiving thiazide therapy should be observed for clinical signs of fluid or ele imbalance, namely: Hyponatremia, hypochloremic alkalosis, and hypokalemia. Serum ar electrolyte determinations are particularly important when the patient is vomiting excess receiving parenteral fluids. Medication such as digitalis may also influence serum elect Warning signs irrespective of cause are: Dryness of mouth, thirst, weakness, lethargy, drow restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycarc gastrointestinal disturbances such as nausea and vomiting.

Hypokalemia may develop, especially with brisk diuresis, when severe cirrhosis is pre during concomitant use of corticosteroids or ACTH.

Interference with adequate oral electrolyte intake will also contribute to hypokalemia kalemia can sensitize or exaggerate the response of the heart to the toxic effect of (eg, increased ventricular irritability). Hypokalemia may be avoided or treated by use of pot supplements, such as foods with a high potassium content.

Any chloride deficit is generally mild and usually does not require specific treatment, under extraordinary circumstances (as in liver or renal disease). Dilutional hyponatremia ma in edematous patients in hot weather; appropriate therapy is water restriction, rather than a tration of salt, except in rare instances when the hyponatremia is life-threatening. In act depletion, appropriate replacement is the therapy of choice.

Hyperuricemia may occur or frank gout may be precipitated in certain patients receiving t therapy.

Insulin requirements in diabetic patients may be increased, decreased, or unchanged. D mellitus which has been latent may become manifest during thiazide administration.

If progressive renal impairment becomes evident, consider withholding or discontinuing therapy.

Thiazides may decrease serum PBI levels without signs of thyroid disturbance.

Calcium excretion is decreased by thiazides. Pathologic changes in the parathyroid gla hypercalcemia and hypophosphatemia have been observed in a few patients on prc thiazide therapy. The common complications of hyperparathyroidism, such as renal lithiasi resorption, and peptic ulceration, have not been seen. Thiazides should be discontinued carrying out tests for parathyroid function.

DRUG INTERACTIONS: Thiazide drugs may increase the responsiveness to tubocurarir

The antihypertensive effects of thiazides may be enhanced in the postsympathectomy Thiazides may decrease arterial responsiveness to norepinephrine. This diminution is not su to preclude effectiveness of the pressor agent for therapeutic use.

PREGNANCY: Pregnancy Category C. Thiazides cross the placental barrier and appear blood. The use of thiazides in pregnancy requires that the anticipated benefit be weighed possible hazards to the fetus. These hazards include fetal or neonatal jaundice, thrombocyt and possibly other adverse reactions which have occurred in the adult.

NURSING MOTHERS: Thiazides appear in human milk. If use of the drug is deemed es the patient should stop nursing.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Propranolol hydrochloride (INDERAL® LA): Most adverse effects have been m transient and have rarely required the withdrawal of therapy.

Cardiovascular: Bradycardia; congestive heart failure; intensification of AV block; hypot paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the R type.

Central Nervous System: Lightheadedness; mental depression manifested by ins lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; disturbances; hallucinations; an acute reversible syndrome characterized by disorienta time and place; short-term memory loss; emotional lability; slightly clouded sensoriu decreased performance on neuropsychometrics.

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, pation; mesenteric arterial thrombosis; ischemic colitis.

Allergic: Pharyngitis and agranulocytosis; erythematous rash; fever combined with ach sore throat; laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis; nonthrombocytopenic purpura, thrombocytopenic purp

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been rep

Miscellaneous: Alopecia; LE-like reactions; psoriasiform rashes; dry eyes; male impoten Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving tl serous membranes, and conjunctivae reported for a beta blocker (practolol) have nc associated with propranolol.

Hydrochlorothiazide:

Gastrointestinal: Anorexia; gastric irritation, nausea, vomiting, cramping, diarrhea, const jaundice (intrahepatic cholestatic jaundice); pancreatitis; sialadenitis.

Central Nervous System: Dizziness, vertigo; paresthesias; headache; xanthopsia.

Hematologic: Leukopenia; agranulocytosis; thrombocytopenia; aplastic anemia.

Cardiovascular: Orthostatic hypotension (may be aggravated by alcohol, barbitura narcotics).

Hypersensitivity: Purpura; photosensitivity; rash; urticaria; necrotizing angiitis (va cutaneous vasculitis); fever; respiratory distress, including pneumonitis; anaphylactic reac

Other: Hyperglycemia; glycosuria; hyperuricemia; muscle spasm; weakness; restles transient blurred vision.

Whenever adverse reactions are moderate or severe, thiazide dosage should be redu therapy withdrawn.

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ECHOCARDIOGRAPHIC SCREENING FOR ACUTE INFECTIOUS ENDOCARDITIS IN DRUG ABUSERS

ALLEN B. WEISSE, M.D., AND MARY CEREBE NOBLE, NEWARK*

In 331 patients, almost exclusively intravenous drug abusers with suspected endocarditis, the results of echocardiography were reviewed. The overall low yield of positive results (26 cases) suggests the need for future study in more efficient management of such patients at risk.

University Hospital of UMDNJ in Newark serves as the acute care facility for an inner-city community where intravenous drug abusers (IVDAs) represent a major health care problem. In the echocardiography laboratory, of the 1,700 examinations performed annually, between 17 and 20 percent, or over 300 such procedures, are performed in IVDAs admitted with the suspicion of acute infectious endocarditis (AIE).

Recent echocardiographic studies in patients shown to have infectious endocarditis by other means, either clinically or at autopsy, have demonstrated positive echocardiographic findings in between 80 and 100 percent of such cases.[1-5] The early detection of vegetations by echocardiography, before the results of blood cultures and other laboratory modalities have established the diagnosis, could allow theoretically for earlier decisions to be made in regard to the institution of appropriate antibiotic and possibly cardiac therapy. With this consideration in mind, we have studied echocardiographically all patients suspected of having AIE on admission to this institution over the last three years.

The current investigation was performed to evaluate the efficacy of this approach to the diagnosis of AIE as well as to compare the relative values of M-mode and two-dimensional echocardiography in regard to this problem, specifically as related to the IVDA population.

PATIENTS AND METHODS

We reviewed the echocardiographic records of all patients referred to this laboratory for possible AIE between November 1981 and May 1984. This included all known or suspected IVDAs admitted to this institution with unexplained fevers, new heart murmurs, or possible evidence of vegetation embolization to the lungs or systemic circulation. Other patients with clinical findings suggestive of infectious endocarditis also were included. We extracted the results of all those studies in which both complete M-mode evaluation (aortic, mitral, and tricuspid valves seen) and 2-D echocardiography were performed successfully. In order to focus on the problem of the IVDA with suspected AIE, we eliminated from further consideration all patients with underlying cardiac valvular abnormalities such as those due to rheumatic heart disease, congenital valve disease, and previous prosthetic valve insertion. There were 331 patients whose studies were analyzed. Of these, over 95 percent were felt unequivocally to be IVDAs.

M-mode tracings were taken with an Irex II system and 2-D echocardiograms with an Advanced Technology Laboratories Mark 300C System utilizing a 2.25

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MHz mechanical scanning head. All tracings were evaluated by the senior author. To avoid any possible influence of the 2-D findings on the interpretation of the M-mode results, the latter were presented first to the reviewer without prior knowledge of the 2-D findings. Valve appearance on the M-mode tracings was judged to be normal, abnormal, or possibly abnormal (the latter when the typical multiple echoes or "shaggy" echoes of vegetations were not present but valve appearances nonetheless raised suspicions of endocarditis).

In order to locate any additional cases of AIE perhaps not referred to the echocardiography laboratory, a search was conducted within the medical records department for the period in question.

RESULTS

Among the 331 cases analyzed, vegetations were detected by 2-D echocardiography in 26 instances among 25 individuals, all but 4 of whom were known IVDAs. Of the nondrug abusers, 1 was a woman undergoing treatment for hypernephroma, 2 were patients with mitral valve prolapse, and 1 patient had a history of rheumatic fever but no prior stigmata of this either on physical examination or echocardiography.

A single valve was involved in 23 cases: 7 aortic, 7 mitral, and 9 tricuspid. In 2 cases, the aortic and mitral valves were involved and in 1 both the mitral and tricuspid valves were affected. The results of the 2-D and M-mode studies are indicated in Table 1. In only 2 cases, 1 involving the mitral valve and 1 the tricuspid, was the M-mode appearance of the involved valve completely normal. No case was judged definitely abnormal on M-mode and not confirmed as such on 2-D examination.

A subsequent review of the charts of those patients with positive findings revealed that, on the basis of bacteriological findings and clinical course, three cases actually constituted instances of subacute rather than acute endocarditis. In two individuals the vegetations turned out to be inactive, i.e. scarred valves from previous episodes of endocarditis. A medical records search conducted for the period of this study revealed no additional cases of acute endocarditis.

DISCUSSION

As with previous reports,[1-5] the current study demonstrates the usefulness of echocardiography i diagnosing vegetations in infectious endocarditi Even the M-mode technique, alone, in a large numbe of such patients will reveal findings that are eithe positive for or suspicious for endocarditis prior to confirmation by 2-D echocardiography. Our results an those of others are compared in Table 2.

The fact that in the present study no additiona cases were uncovered by a medical records searc underlines the usefulness of this technique in th present-day diagnosis of infectious endocarditis.

The major finding of the current study is that so fe of the IVDAs admitted with fevers and suspected endocarditis actually turned out to have this disease. C all patients with suspected endocarditis evaluated an who underwent both M-mode and 2-D studies, ther were positive results in only 1 of every 13 patients, i. acute, healed, or subacute endocarditis. If one adde in the many cases who had clearly normal M-mod studies and therefore no 2-D evaluations, the yiel would be even smaller. Should AIE have been misse initially by M-mode, given the housestaff's acut awareness of this entity and the importance o echocardiography, such cases would, undoubtedl have been referred back to the laboratory.

The possible causes for fever in the IVDA populatio are several, including infectious endocarditis. Ther may be bacterial or viral infection not involving th heart valves. There also may be febrile reactions t illicitly used drugs or their vehicles.

TABLE 1

Echocardiographic Detection of Vegetations by M-Mode and 2-D Techniques

		Aortic	Mitral	Tricuspid
2-D		9	10	10
M-mode:	**positive**	8	4	6
	possible	1	5	3
	normal	0	1	1

TABLE 2

Comparison of Two-Dimensional and M-Mode Findings in Endocarditis

		Aortic Value		Mitral Valve		Tricuspid Valve
	2-D	M-Mode	2-D	M-Mode	2-D	M-Mode
Mintz et al., 1979[7]	10	7 (3)*	4	4(3)	5	4
Wann et al., 1979[8]	8	8	7	7	3	3
Berger et al., 1980[2]	-	-	-	-	10	5
Berger et al., 1981[4]	12	8	-	-	-	-
Ginzton et al., 1982[9]	-	-	-	-	16	12 (2)
O'Brien et al., 1983[10]	6	3	9	8	3	2
Current Study	9	9 (1)	10	9 (5)	10	9 (3)
Total	45	35 (4)	30	28 (8)	47	35 (5)

*The figure in parentheses indicates the number of M-mode studies (included in the total) where tracings were considered either suspicious or suggestive of vegetations.

Even when bacteremia is present this may be related to infection elsewhere in the body. Pertinent to this is a previous study of bacteremia in a number of patients with underlying heart disease predisposing to infectious endocarditis.[6] Among 57 cases with bacteremia, only 9 cases were shown to have infectious endocarditis on echocardiography. One additional case with an abnormal mitral valve on echocardiography not called "endocarditis" was proved at autopsy.

The results of this study suggest avenues of investigation requiring further attention. To begin with, ultrasonic evaluation of IVDAs for AIE represent a major investment of time, effort, and dollars at institutions such as University Hospital. But hospitalization is even more expensive. Among this group, echocardiography will show normal results in the large majority of such referrals. Which febrile IVDAs who show no echocardiographic evidence of endocarditis will require admission to hospital? Perhaps in certain subgroups, e.g. those with no obvious source of infection, normal chest films, and no evidence of systemic embolization, the echocardiogram might be useful in identifying IVDAs who safely can be managed as outpatients in view of the great likelihood that no serious infection exists.

The development of future guidelines for the more efficient use of hospital and laboratory resources now is under active consideration and study at this institution.

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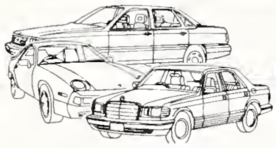

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Case Report: Cystosarcoma Phyllodes with Liposarcomatous Stroma in the Female Breast

Leonard Galler, M.D., and Robert J. Beach, M.D., Somers Point*

Cystosarcoma phyllodes with liposarcomatous stroma in the female breast are a very rare malignancy. Our case report documents the fact that liposarcoma can arise de novo as well as in association with other breast tumors, such as malignant cystosarcoma phyllodes.

Sarcomas of the breast constitute a 0.5 to 3 percent of all malignant breast neoplasms.[1,2] Within this group, cystosarcoma phyllodes are the most frequently encountered. Liposarcoma of the breast, unlike cystosarcoma phyllodes, is an extremely rare and poorly understood malignancy, representing 0.3 percent of all breast sarcomas.[3] Liposarcoma may arise de novo or from pre-existing breast tumors, such as malignant cystosarcoma phyllodes.[4-6] Neumann first described liposarcoma of the breast in 1862.[7] Subsequent literature shows 51 cases of liposarcoma of the breast. These cases primarily arose as pure liposarcoma, but several were associated with other breast tumors. Because of the rarity of this neoplasm, questions arise regarding its malignant potential, clinical presentation, and methods of treatment.

We report the case of our patient who had cystosarcoma phyllodes with a liposarcomatous stroma arising in the female breast. Histological features, clinical presentation, and therapy will be discussed. Comparisons with other previously reported cases will be used to help elucidate this neoplasm and establish guidelines for treatment.

CASE REPORT

A 59-year-old healthy white female developed a rapidly enlarging, painless right breast mass occurring over several months. She was postmenopausal, took no medications, and had a negative family history for breast malignancies.

Physical examination was negative, except for a firm, nonfluctuant, nontender, subareolar right breast mass measuring 4 cm x 4 cm x 5 cm. Both breasts were large and pendulous. The mass was quite mobile without skin retraction or nipple involvement. Axillary examination was negative. Chest x-ray and bone scan were negative. Laboratory studies, including liver function tests, were normal.

Mammography did demonstrate the right breast mass; there were no calcifications seen. The stroma pattern on the left breast was normal except for some left upper-quadrant benign calcifications.

Needle aspiration, as an office procedure, showed numerous histiocytes, but no malignant cells. Outpatient excisional biopsy demonstrated a low-grade myxoid liposarcoma. A right simple mastectomy was performed subsequently. The residual tumor in the mastectomy specimen was malignant cystosarcoma phyllodes with a predominantly myxoid liposarcomatous stroma (Figure). All margins of the specimen were free of tumor.

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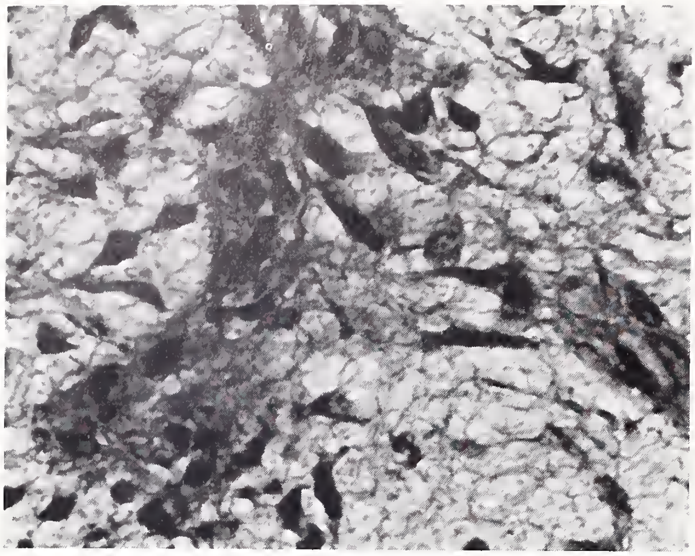
The six-month and one-year followup visits revealed no evidence of local recurrence or distal metastases. Repeat chest x-ray and liver function tests were negative. Repeat mammography of the left breast one year later demonstrated stippled calcifications; subsequent outpatient biopsy of this area showed benign disease. No adjuvant treatment was given.



Figure— Myxoid liposarcomatous stroma with spindle to stellate hyperchromataic nuclei and vacuolated cytoplasm (hematoxylin-eosin x 400).

PATHOLOGY

The gross appearance of the initial biopsy specimen demonstrated five irregular tissue fragments which ranged in size from 3.5 cm to 1.2 cm in greatest dimension. Each fragment contained well-circumscribed areas of homogenous, gelatinous, translucent, gray-yellow tumor with adjacent white and yellow breast parenchyma. At the time of the simple right mastectomy, the remaining tumor mass measured 4.5 cm x 3.5 cm x 3.5 cm, and was composed of soft, lobulated, gelatinous, translucent, gray-yellow focally hemorrhagic tissue. It appeared well-circumscribed immediately beneath, but not involving the skin. There was no gross evidence of multifocality of the tumor with negative widely resected margins.

Histologically, the biopsy demonstrated well-circumscribed areas of myxomatous-appearing tumor with a prominent, delicate, branching (chicken wire-like) vascular pattern. Lipoblasts characteristic of liposarcoma were present; they had stellate nuclei with rare multinucleated tumor giant cells. Marked vacuolation of the cytoplasm was noted throughout, with slightly vacuolated nucleated chromatin. No mitotic figures were identified. Rare normal appearing breast ducts were present within the tumor, primarily at the tumor margins. The tumor extended into the surrounding breast parenchyma with a pushing margin, and with no microscopic evidence of a distinct fibrous capsule.

Microscopic examination of the resected residual tumor demonstrated a similar histologic appearance with two additional findings. Focal large irregular elongated cystic areas within the tumor were lined by one to two layers of low cuboidal epithelium; adjacent areas with microscopic foci of highly cellular stroma demonstrated cystosarcoma phyllodes. These areas represented less than 5 percent of the tumor mass. The tumor again demonstrated pushing margins without a distinct capsule. Random sections of the remaining breast tissue demonstrated benign intraductal papillomatosis with no evidence of multifocality of the tumor.

The gross and microscopic appearance of the initial biopsy specimen were entirely consistent with a classic low-grade myxoid liposarcoma originating in the breast, with the highly vascular stroma and numerous lipoblasts suggesting that diagnosis. Fat stains and ultrastructural studies were not performed on this specimen because of the typical appearance of the lipoblasts on light microscopy. These findings were confirmed by a consultant. Examination of the residual tumor within the mastectomy specimen, however, indicated the true nature of the lesion as malignant cystosarcoma phyllodes with a predominant myxoid liposarcomatous stromal component. Similar lesions have been reported by Oberman and Quizilbash.[3,6]

DISCUSSION

Liposarcoma of the breast can behave in a very aggressive malignant fashion with rapid local and distant metastases or in a less virulent fashion. Neumann and Geschichter described cases of liposarcoma of the breast which metastasized rapidly and killed the patient, often due to hematogenous spread to the lungs.[7,8]

Lymph node involvement was less frequent. Histological patterns often depicted undifferentiated mucoid liposarcoma with many bizarre lipoblasts varying in size, shape, and polarity. This pattern corresponds to the poorly undifferentiated types described by Enterline who suggested five histological types of liposarcoma: I) well-differentiated myxoid; II) poorly differentiated myxoid; 3) lipoma-like; IV) mixed lipoid type; and V) nonmyxoid type.[9,18] The more undifferentiated and the nonmyxoid types were most often noted to metastasize.[9]

Liposarcomas of the breast can occur at any age (16 to 76), but usually occur in women ages 40 to 60.[11] Only one male has been reported with this lesion.[12] The usual complaint is a single breast mass, but two patients, including a lactating female, had bilateral liposarcoma.[11,13]

Although early reports advocate radical mastectomy as definitive treatment for liposarcoma of the breast, current appropriate treatment is simple mastectomy, removing all tumor. Larger lesions, with poorer histological patterns, require more radical surgery to obtain clear margins. Axillary lymph nodes and underlying pectoralis muscle should be excised if involved. Our patient's isolated singular breast mass not involving the axilla was adequately treated by simple mastectomy. The effect of radiation therapy and chemotherapy is unknown. Although liposarcomas are relatively radioresistant, Geschichter and Stout advocate its use in advanced disease or as a palliative step, but the benefit of adjuvant chemotherapy still is to be assessed.

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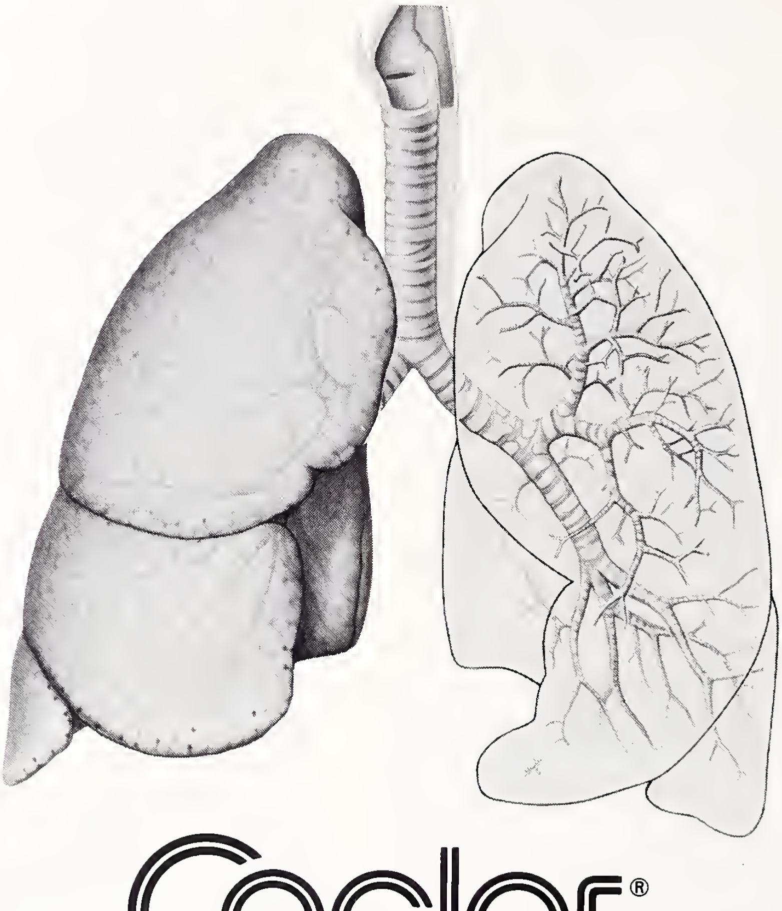

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Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out

Precautions: *General Precautions* — If an allergic reaction to Ceclor® (cefaclor, Lilly) occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly)

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Usage in Pregnancy — Pregnancy Category B — Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor® (cefaclor, Lilly). There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers — Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor is administered to a nursing woman.

Usage in Children — Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below:

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbiliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vagin (less than 1 in 100 patients).

Causal Relationship Uncertain — Transitory abnormalitie clinical laboratory test results have been reported. Although were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic — Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic — Transient fluctuations in leukocyte cour predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal — Slight elevations in BUN or serum creatinine (less 1 in 500) or abnormal urinalysis (less than 1 in 200).

(0617

Note: Ceclor® (cefaclor, Lilly) is contraindicated in patients with known allergy to the cephalosporins and should be give cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophyl of rheumatic fever. See prescribing information.

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CASE REPORT: BILATERAL PNEUMOTHORAX, A RARE COMPLICATION OF SKIN POPPING

RAVINDRA I. PATEL, M.D., AND SHASHI K. AGARWAL, M.D., EAST ORANGE*

A rare case of self-induced bilateral pneumothorax is reported. Intravenous drug abuse should be suspected as the etiology, especially when it is bilateral. Inept attempts at injecting in large neck veins lacerates the pleura. Prompt surgery may be necessary.

Pneumothorax, which occurs in approximately 0.2 percent of all hospital admissions,[1] is associated with lung diseases,[2-5] but may occur spontaneously.[6-8] The development of critical care intervention has added another major etiology. Pneumothorax is documented in 4.2 percent of patients on mechanical ventilation.[9] It has followed central venous line catheterization,[10] endotracheal or esophageal intubation,[11] transtracheal aspiration,[12] chemotherapy,[13] radiation therapy,[14] and acupuncture.[15] Intravenous drug abuse has introduced another cause for pneumothorax as a complication of internal jugular vein injection.[11-18] The production of bilateral pneumothorax due to "skin popping" is extremely rare, with only one case noted in the recent medical literature.[19]

We report another case of self-induced bilateral pneumothorax in a 33-year-old female to draw attention to this potentially lethal complication associated with the illicit use of drugs.

CASE REPORT

A 33-year-old female came to the emergency room at United Presbyterian Hospital with complaints of chest pain and shortness of breath. The symptoms started approximately ten minutes following unsuccessful attempts to inject "speed ball" (cocaine and heroin), into either internal or external jugular veins. There was no history of heart or lung disease, but there was a history of hepatitis. She had hypertension for which she was taking propranolol 60 mg by mouth daily and furosemide 40 mg twice a week. She denied gastrointestinal, genitourinary, or central nervous system disease. She smoked about two packs of cigarettes per day, was an alcoholic, and was a drug abuser for 14 years. She was enrolled in a methadone detoxification program at the time of this illness.

The patient was afebrile but dyspneic with a respiratory rate of 38/min. Blood pressure was 140/110 mm Hg; pulse was 104/min. Scars at numerous injection sites were on the extremities and both sides of the neck. We noted poor inspiratory expansion with resonance to percussion on both sides (R>L). PMI was not localized but S1 and S2 were normal. The abdomen and neurological systems were unremarkable. EKG revealed low voltage. Chest x-ray revealed bilateral pneumothorax with complete collapse of the right lung and 30 percent collapse of the left lung. Arterial blood gases revealed Ph 7.46; $PC0_2$ 36 mm Hg; $P0_2$ 64 mm Hg; and $HC0_3$ 25 mEq/L on room air.

The patient was treated with bilateral chest tube insertions and the pneumothoraces gradually resolved.

---

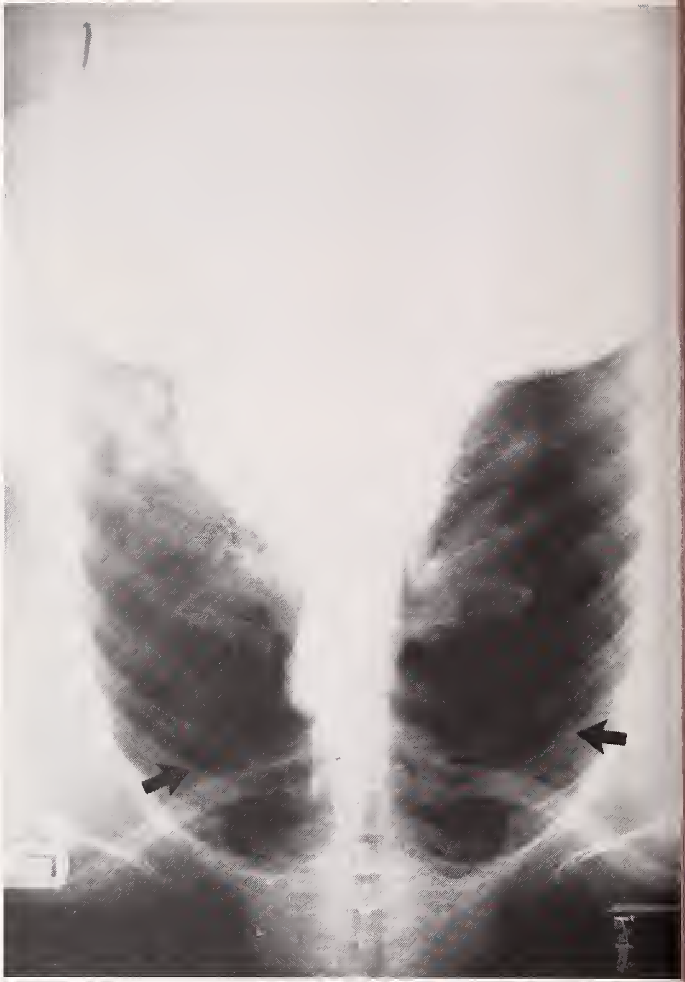
*Dr. Patel is affiliated with the Department of Internal Medicine, and Dr. Agarwal is affiliated with the Division of Cardiology, United Hospitals Medical Center, Newark. Correspondence may be addressed to Dr. Agarwal, 144 South Harrison Street, Suite 502, East Orange, NJ 07018.

DISCUSSION

Intravenous drug abuse in the United States is on the rise.[20] This has resulted in a number of serious complications such as endocarditis,[21] hepatitis,[22] and possibly AIDS.[23] Long-term intravenous drug abuse gradually obliterates peripheral veins by an infectious or sclerotic process.[24,25] The addict then will turn to larger veins such as the internal jugular vein. The proximity of several vital structures in this area and the limited skill of the "skin popper" subjects the addict to the risk of significant morbidity and mortality.[17] Local complications include abscesses and/or cellulitis, internal jugular vein thrombosis, and mycotic aneurysm as well as severe neurologic injuries.[17,19,26]

Access to the internal jugular vein usually is obtained by injection into the depression in the neck lateral to the sternocleidomastoid muscle immediately above the clavicle ("the pocket shot").[19] Since the apical pleura lies adjacent to this access site, even slight lateral angulation of the needle may result in laceration of this structure and consequent pneumothorax.

Our patient was unusual in that she developed bilateral pneumothoraces. The failure to enter the internal jugular vein on the left side prompted a right-sided approach resulting in bilateral pleural laceration. Prompt medical and surgical therapy obviated a potential catastrophe.



Figure—Chest x-ray revealing bilateral pneumothorax (arrows).

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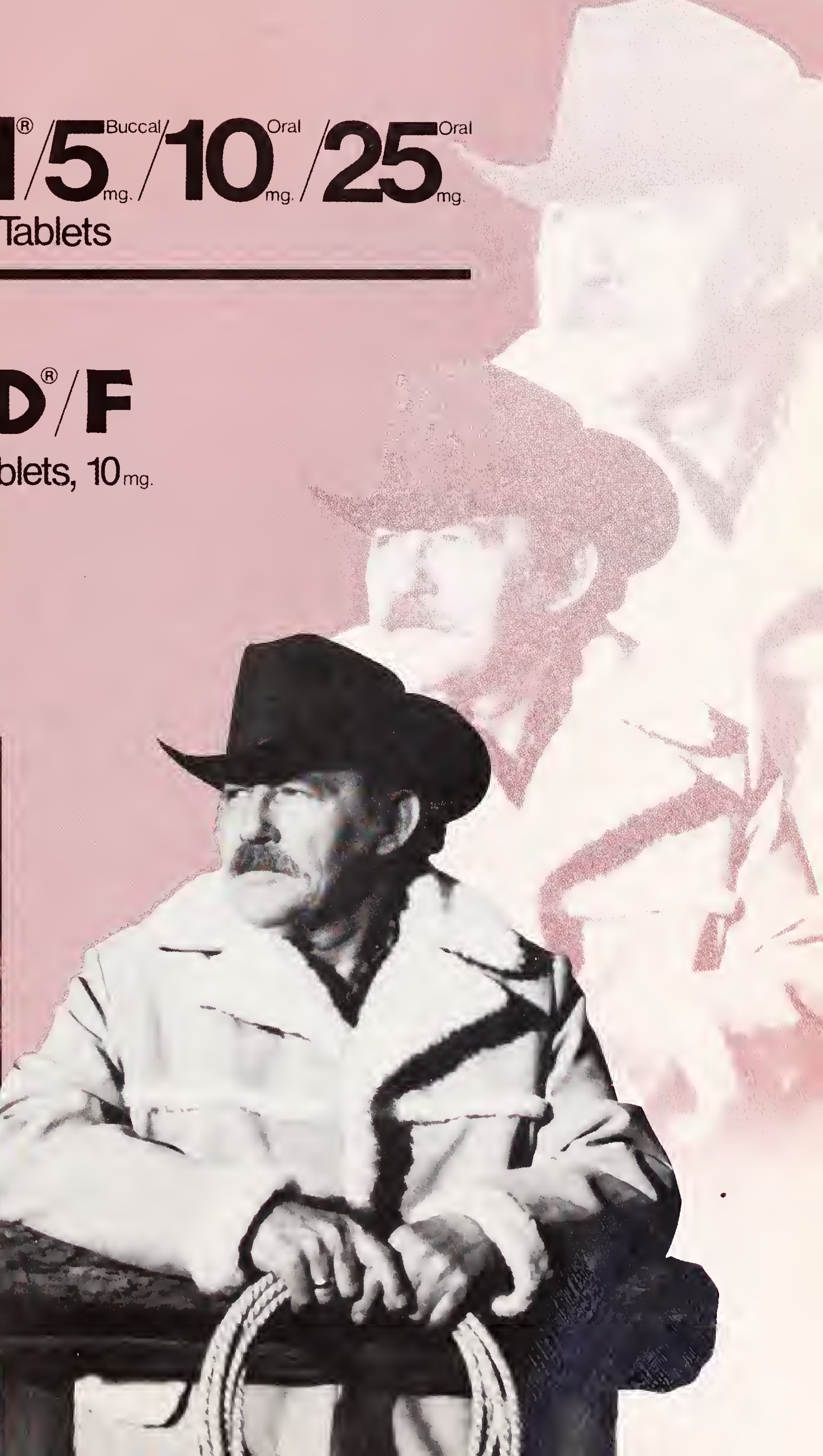

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REPORT OF THE NOMINATING COMMITTEE

Offices To Be Filled By Election—1986 Annual Meeting

Office	**Term**	**Nominee and County**
President-Elect	1 year	Harry M. Carnes, M.D., Camden
1st Vice-President	1 year	Palma E. Formica, M.D., Middlesex
2nd Vice-President	1 year	Paul J. Hirsch, M.D., Somerset
Secretary	3 years	Bernard Robins, M.D., Essex
Treasurer	3 years	Douglas M. Costabile, M.D., Union
Trustees		
1st District	3 years	Joel S. Cherashore, M.D., Essex
2nd District	3 years	Frank Gingerelli, M.D., Bergen
5th District	3 years	John J. Pastore, M.D., Cumberland
Judicial Councilors		
2nd District	3 years	Alden B. Hall, M.D., Sussex
5th District	3 years	Paul H. Steel, M.D., Atlantic
AMA Delegates		
	2 years	Alfred A. Alessi, M.D., Bergen
	2 years	Frederick W. Durham, M.D., Camden
	2 years	Palma E. Formica, M.D., Middlesex
	2 years	Karl T. Franzoni, M.D., Mercer
	2 years	John S. Madara, M.D., Salem
	2 years	Henry J. Mineur, M.D., Union
AMA Alternate Delegates		
	1 year	James H. Spillane, M.D., Warren
	2 years	Joseph N. Micale, M.D., Hudson
	2 years	James H. Spillane, M.D., Warren
	2 years	Michael M. Heeg, M.D., Mercer
	2 years	Charles S. Krueger, M.D., Burlington
	2 years	Joseph A. Riggs, M.D., Camden
	2 years	Robert H. Stackpole, M.D., Union
Administrative Councils		
Legislation		
1st District	2 years	Anthony P. Caggiano, Jr., M.D., Essex
4th District	2 years	Irving P. Ratner, M.D., Burlington
Medical Services		
1st District	2 years	Albert Minzter, M.D., Union
4th District	2 years	Lindsay L. Pratt, M.D., Camden
Mental Health		
1st District	2 years	Rita R. Newman, M.D., Essex
2nd District	2 years	William H. Bristow, Jr., M.D., Bergen
Public Health		
1st District	2 years	Richard R. Lorber, M.D., Union
4th District	2 years	William Pawluk, M.D., Burlington
Public Relations		
1st District	2 years	Charles H. Arnoldi, Jr., M.D., Essex
4th District	2 years	Blackwell Sawyer, Jr., M.D., Ocean
Standing Committees		
Annual Meeting	2 years	John F. Marshall, M.D., Mercer
Auxiliary Advisory	2 years	No nominee
Finance and Budget	2 years	B. Ralph Wayman, Jr., M.D., Mercer
Medical Education	2 years	Roberta G. Rubin, M.D., Passaic
Membership Services	2 years	Carl Restivo, Jr., M.D., Hudson
Publication	2 years	Frederick B. Cohen, M.D., Essex

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DOCTORS' NOTEBOOK

Trustees' Minutes; UMDNJ Notes; MSNJ Auxiliary; New Members; Widows and Orphans Society Meeting; Physicians Seeking Location in New Jersey

Trustees' Minutes February 16, 1986

A regular meeting of the Board of Trustees was held on Sunday, February 16, 1986, at the Executive Offices in Lawrenceville. Detailed minutes are on file with the secretary of your county society. A summary of significant actions follows:

Armando F. Goracci, M.D. . . . Observed a moment of silence in tribute to the memory of Dr. Goracci who died February 7, 1986. Noted that memorial contributions were made to the Alumni Office, Jefferson Medical College, and MSNJ's Student Loan Fund in memory of Dr. Goracci.

AIDS—Union County Medical Society's Resolutions . . . Referred to the Council on Public Health for consideration and report back to the Board, resolutions concerning AIDS and AIDS treatment in New Jersey.

Report of the President . . .
(1) Regionalized Society Services . . . Noted Dr. Fioretti's concern for more input from county societies on MSNJ services.
(2) Conference on HMOs . . . Commended Bernard Robins, M.D., for his outstanding program on HMOs, PPOs, and alternative delivery systems.

Executive Committee . . .
(1) Quality of Medical Records . . . Agreed that the concern for the quality of medical records should be presented to the Governing Council of the Hospital Medical Staff Section (MSNJ) and Medical Administrators Committee (NJHA) for study and recommendations.
(2) Uncompensated Care Legislation . . . Noted the NJHA proposed bill establishing a trust fund to pay for all charity care and bad debts to New Jersey's acute, nongovernmental hospitals; monies would come from a levy on payor, liquor, and cigarette taxes; and state funding of Medicaid participation in the fund. Presented this proposal to the Council on Legislation for recommendations.
(3) Proposed Amendments—Utilization Review Organizations . . . Submitted comments on proposed amendments and noted NJHA support for the MSNJ position.
(4) Task Force Reports on Capital and Quality . . . Noted objections to the conclusions of the Department of Health's Task Force Reports on Capital and Quality and will draft a statement with NJHA for the governor and the Health Care Administration Board.
(5) Joint Ventures . . . Adopted the following resolution:

Whereas, joint ventures by hospitals and their medical staffs are essential to the survival of private practice and the voluntary hospital system; and

Whereas, there has been frequent discussion but little substantive information regarding the practicality of joint ventures in New Jersey; now therefore be it

Resolved, that the Medical Society of New Jersey and the New Jersey Hospital Association jointly produce a regional seminar on the status of practicality of joint hospital-medical staff ventures in New Jersey.

Report of Executive Director . . .
(1) MSNJ Membership . . .
(a) Paid 1985 Membership . . . Noted that as of December 31, 1985, paid membership was 7,574.
(b) Paid 1986 Membership . . . Noted that as of February 14, 1986, paid membership was 5,589.
(2) Additional Office Space and Renovations . . . Approved the following recommendations.

(a) That the headquarters building be altered to provide an additional 13,000+ square feet of space for the use of the New Jersey State Medical Underwriters, Inc. (NJSMU).

(b) This alteration will involve a conversion of 50 percent of the delegates' room to office space for the New Jersey State Medical Underwriters, Inc. Fifty percent of the room will be redesigned for conference and dining purposes. Sixty percent of the current warehouse area will be double-decked and converted to NJSMU leasehold.

(c) Financing is to be arranged through a loan from the Medical Inter-Insurance Exchange of New Jersey.

(d) Loan amortization, plus a modest return, are to be met through the rental for the new area.

(3) Repeal of Certificate of Need Law . . . Approved the following recommendation:

Based on the results of the survey of New Jersey hospitals, the Council on Legislation and the House of Delegates should be advised that presently it is not feasible to pursue a legislative campaign for general repeal of the certificate of need law; but that the Medical Society of New Jersey should maintain its opposition to the certificate of need law, both generically and specifically, as it relates to private practice.

(4) MSNJ Financial Statements . . . Approved the financial statements for the period ending December 31, 1985, and January 31, 1986.
(5) Committee on Membership Services . . . Granted permission to the Committee to extend an invitation to the Princeton Insurance Company to present an outline of their insurance program for physicians.

NJ Hospital Association . . .
(1) Medicare . . . Noted that New Jersey hospitals are being asked to join a campaign to have Congress maintain at least the 1 percent increase for Medicare provided in the original Deficit Reduction Act.
(2) HMO Project . . . Noted that NJHA President, Mr. Scibetta, agreed to inform the Board of all significant developments regarding the HMO project.

Council on Mental Health . . . Adopted the following amended recommendation:

That the New Jersey Academy of Family Physicians be advised that their resolution dealing with the insanity defense in New Jersey is not in concert with the views subsequently developed and approved by the American Medical Association, the American Psychiatric Association, and the New Jersey Psychiatric Association, and, therefore, should be reconsidered.

Peer Review Oversight Committee . . .
(1) Representative at PRO Criteria Committee Meetings . . . Accepted the following recommendation with the proviso that acceptance of PRO's invitation to send an observer in no way diminishes or supplants the Board's prior request to send a representative to The Peer Review Organization of New Jersey, Inc., board meetings:

That the Board of Trustees be asked to accept the invitation of The Peer Review Organization of New Jersey, Inc., to send a member of the Peer Review Oversight Committee to PRO's Criteria Committee meetings as an observer only. (Any member of the PRO Criteria Committee sent to Peer Review Oversight Committee meetings would likewise have observer status only.)

(2) Advising the Patient of Denial . . . Approved and referred to the AMA Delegation the following recommendation:

That the Board of Trustees be asked to introduce a resolution requiring the AMA to go before the Health Care Financing Administration for review of the current system of denial notification to the patient—made by PRO via letter, as late as one year after the hospital stay. (A better idea would be for PRO to inform the patient verbally within 30 days of his/her denial, assuming responsibility for the decision and answering the patient's questions.)

Ad Hoc Committee on Health Care Reimbursement Policies . . .
(1) Preparing Physicians for Reimbursement Changes . . . Voted to postpone consideration of the following recommendation:

That the Board of Trustees embark on the issuance of a periodical to be mailed separately from *NEW JERSEY MEDICINE* and the *Membership Newsletter* contained therein, on the socioeconomic issues facing physicians today, including an editorial comment by the Board of Trustees on how the practice of medicine in New Jersey will be affected.

(2) Inappropriateness of DRG Remuneration—1985 House of Delegates . . . Noted that MSNJ will continue to monitor the DRG program.

Committee on Drug and Alcohol Abuse . . . Approved to following recommendations:

That the Board of Trustees approve the Committee's position that the need for removal of over-the-counter drugs from self-service areas is not indicated at the present time.

Old Business . . .
(1) Petition for House of Delegates Seating . . . Voted to postpone consideration of the petition from the Society of Surgeons of New Jersey requesting seating in the MSNJ House of Delegates.

(2) Second-Level Appeals—New Jersey DRG Program . . . Voted to maintain MSNJ position not to designate two physician representatives for the committee being restructured to hear second-level appeals.

(3) Future Annual Meetings . . . Referred the matter of future annual meetings to the Committee on Annual Meeting. Also, noted the new time and location of the 1986 Annual Meeting is April 30 to May 4 at the Host Farm Resort.

New Business . . .
Physicians' Union . . . Invited a delegation of Mercer County physicians to submit a written statement on the subject of a physicians' union.

UMDNJ Notes

Stanley S. Bergen, Jr., M.D.
President

From time to time, I spotlight research underway at UMDNJ and its affiliate hospitals. These investigations range from basic science to clinical, but the goal is one and the same: to lay the groundwork for better patient care.

One such study shows that standard room lighting significantly can extend the lives of hamsters with heart disease. We already know that light has been shown to be therapeutic for some patients suffering from winter depression, but it would indeed be remarkable if light proved to have positive biological effects as well.

The research, conducted over the past four years at the Veterans Administration Medical Center in East Orange by Walter Tapp, Ph.D., and Benjamin Natelson, M.D., of the department of neurosciences at UMDNJ-New Jersey Medical School, showed that the hamsters survived 25 percent longer when exposed to continuous fluorescent light, 24 hours a day, compared to a control group of carefully matched counterparts whose days were equally divided between light and darkness. For the study, Drs. Tapp and Natelson used a breed of hamster with an inherited heart condition that limits its lifespan to about a year, or half that of normal hamsters. There was, therefore, a manageable time frame to study the successive life cycles of hamster populations. The project is titled "Life Extension in Heart Disease—An Animal Model."

"Constant light retarded the progress of the heart failure and also made hamsters living in constant light more likely to survive the early stages of heart failure," the researchers report. "This observation may open new avenues for the treatment of human beings with heart disease."

The doctors added that they are pursuing this line of study to determine if altering an environmental condition—in this case, light—can affect health.

Equally important in the study was the disease itself, congestive heart failure, because both men and hamsters suffer the malady, and in similar forms.

The research consisted of a series of experiments, in which conditions for both groups were duplicated as exactly as possible; it showed that the hamsters in the all-light group lived an average of a little more than 25 percent longer than the hamsters in alternating light and dark, with all the animals given identical access to tap water, nutritious food, and exercise. Another facet of the research included gauging the quality of life for those living the extended terms. The researchers performed autopsies after the hamsters had lived 11 months, so that the cardiovascular systems could be examined. The results: there was much less heart failure in the all-light group.

Dr. Tapp points out that this is significant because it means the disease process was slowed, not that the animals just lived longer in the throes of heart failure.

According to Drs. Natelson and Tapp, there are three possible explanations for the longevity of the hamsters exposed to constant light: that the light itself had a positive biological effect; that the light, representing a longer period of daylight, such as in summertime, had the effect, as expected in the cases of wintertime depression; or that the steady light condition allowed the animals to function on their own circadian rhythms, which served them better than natural time cues.

To understand better the cause and effect, the researchers will continue their studies, using differing light schedules.

Although not anticipating immediate practical application of their findings, the UMDNJ-New Jersey Medical School faculty members added that the model opens many possibilities. For instance, is hospital lighting optimal for patient recovery? Or, what effect does light have on shift workers and others who do not work "typical" hours?

MSNJ Auxiliary

Bernardine Moloney
President

Who does the cooking in your household? I am referring to the extra special dishes that family and friends would love to know how to duplicate.

If you would share one prized recipe with us, it will be included in a cookbook with recipes from the medical families of New Jersey. This cookbook will be published through the joint effort of the Impaired Physician's Committee and the Medical Society of New Jersey Auxiliary. Proceeds from the sale of the cookbook will go to the Treatment Loan Fund of the Impaired Physician's Committee. This fund provides necessary money for a physician and/or family during the time of treatment when there is no other available source of income.

Please help us to produce a book we all can enjoy and take pride in. Send your typewritten recipes to: MSNJ Auxiliary, Two Princess Road, Lawrenceville, NJ 08648.

As Annual Meeting approaches, it is a pleasure to invite every physician's spouse, member, or nonmember to attend the Medical Society of New Jersey Auxiliary's General Session on Saturday, May 3, 1986, 9:00 A.M., and the luncheon immediately following. Attendance at these functions will provide spouses with valuable information on what the Medical Society of New Jersey Auxiliary is accomplishing in its attempts to educate its members and the community about the professional liability crisis, cancer screening, child abuse, and other similar topics.

The keynote speaker at the General Session will be AMA Auxiliary President, Mary Kay McPhee. The program at the luncheon will feature Peter Sweetland, President, MIIENJ (the physician-owned insurance company), and Rev. Edward Reading, Assistant Director, Impaired Physicians Program of the Medical Society of New Jersey. Their topic will be: "The Emotional and Financial Impact of Malpractice Suits."

220th MSNJ ANNUAL MEETING

PROPOSED DAILY SCHEDULE
May 1-4, 1986
Americana Host Farm Resort
Lancaster, Pennsylvania

Wednesday, April 30, 1986
3:30 P.M. —Board of Trustees' meeting

Thursday, May 1, 1986
7:30 A.M. —Registration open
9:00 A.M. —Message Center and Exhibits open
2:00 P.M. —House of Delegates
4:00 P.M. —Reference Committees

Friday, May 2, 1986
8:00 A.M. —Registration, Message Center, and Exhibits open
9:00 A.M. —House of Delegates (election)
12:00 NOON —Golden Merit Award Ceremony followed by Reception
2:30 P.M. —Reference Committees
5:00 P.M. —JEMPAC Political Forum
5:45 P.M. —JEMPAC Wine & Cheese Reception

Saturday, May 3, 1986
8:00 A.M. —Registration, Message Center, and Exhibits open
8:30 A.M. —House of Delegates
1:30 P.M. —House of Delegates
6:30 P.M. —Inaugural Reception followed by Inaugural Dinner

Sunday, May 4, 1986
8:00 A.M. —Registration and Message Center open
8:30 A.M. —General Session—"Corporate Invasion in the Practice of Medicine"
1:00 P.M. —Board of Trustees' meeting

New Members

NEW JERSEY MEDICINE would like to welcome the following new members to the Medical Society of New Jersey:

Bergen County

Carmen Chinea, M.D., Fairview
Leon N. Costa, M.D., Teaneck

Cumberland County

Godofredo R. Bocobo, M.D., Bridgeton

Essex County

Richard J. Bardowell, M.D., East Orange
Myron E. Brazin, M.D., Rutherford
Craig N. Eichner, M.D., West Caldwell
Howard J. Fisher, M.D., Newark
Charles M. Gelber, M.D., Livingston
John J. Gribbon, M.D., Montclair
Joel E. Kopelman, M.D., Livingston
Paul J. LoVerme, M.D., East Orange
Richard V. McCloskey, M.D., Nutley
Rajendra R. Raval, M.D., Newark
Dan S. Reiner, M.D., Newark
William A. Tansey, III, M.D., Short Hills
Frances Yung-Tao Wu, M.D., Verona
Sitamahalakshmi Yerramalli, M.D., Irvington

Hudson County

Muhammad R. Chaudhry, M.D., Jersey City
Anthony G. Gambrino, M.D., Jersey City
Juan A. Gonzalez, M.D., Union City
Michael G. Kailas, M.D., Bayonne

Mercer County

Jerry Bagel, M.D., Hightstown
John C. Baumann, M.D., Princeton
Michael Gersten, M.D., Lawrenceville
Vinod K. Gupta, M.D., Trenton
Amarish R. Sheth, M.D., Trenton
Michael H. Yamane, M.D., Trenton

Middlesex County

Ellen J. Colodney, M.D., Somerset

Monmouth County

James P. O'Neill, M.D., Neptune
Rocco F. Pascucci, M.D., Holmdel

Morris County

John S. Banas, M.D., Morristown

Ocean County

Moshe Bacharach, M.D., Lakewood
Amelia J. Cardo, M.D., Toms River
Lubmilla Engelbach, M.D., Toms River
Narciso D. Mendoza, M.D., Barnegat
James E. Pancurak, M.D., Point Pleasant
Kutumba S. Pitta, M.D., Toms River

Passaic County

Kazem K. Behnam, M.D., Haledon

Union County

Joel S. Bogner, M.D., Cranford

Warren County

Frank J. Gilly, Jr., M.D., Washington
Kee S. Kim, M.D., Washington

ARE YOU MOVING?

If so, please send a change of address to *NEW JERSEY MEDICINE*, Medical Society of New Jersey, Two Princess Road, Lawrenceville, NJ 08648, at least six weeks before you move.

Meeting of Society for Relief of Widows and Orphans

The Annual Meeting of the Society for the Relief of the Widows and Orphans of Medical Men of New Jersey will be held on Saturday, May 3, 1986, at 3:30 P.M. at the Americana Host Farm Resort, Wood Room, Lancaster, Pennsylvania, in conjunction with the Annual Meeting of the Medical Society of New Jersey.

Physicians Seeking Location in New Jersey

The following physicians have written to the Executive Offices of MSNJ seeking information on possible opportunities for practice in New Jersey. The information listed below has been supplied by the physician. If you are interested in any further information concerning these physicians, we suggest you make inquiries directly to them.

ANESTHESIOLOGY—Yellagondahalli V. Janardhan, M.D., 18 Agatha Dr., Edison, NJ 08817. Bangalore (India) 1971. Board certified. Group, partnership, fee for service. Available.

EMERGENCY MEDICINE—Julian A. Sellek, M.D., 1501 S. Greenway, Coral Gables, FL 33134. Salamanca (Spain) 1963. Also, family practice. Board eligible. Board certified (FP). Group, solo, partnership. Available.

FAMILY PRACTICE—Lawrence S. Gordon, M.D., 1016 Spinnaker Way, Apt. G-1, Melbourne, FL 32935. Rutgers 1982. Board certified. Group, partnership, solo. Available.

Natalia Moishezon, M.D., 207 Christie St., Leonia, NJ 07605. Moscow 1964. Board eligible. Group, partnership, industry. Available.

Jeffrey C. Schultz, M.D., 301 Victoria Dr., Monroeville, PA 15146. University of Massachusetts 1983. Board eligible. Group. Available July 1986.

Julian A. Sellek, M.D., 1501 S. Greenway, Coral Gables, FL 33134. Salamanca (Spain) 1963. Also, emergency medicine. Board certified. Board eligible (EMER). Group, solo, partnership. Available.

Lew Herbert Valliant, M.D., 115 Mountain View Dr., Elkins, WV 26241. Queen's University (Canada) 1976. Board certified. Solo or group. Available.

GASTROENTEROLOGY—Richard J. Fastiggi, M.D., 10 Silver Stirrup Ct., Timonium, MD 21093. UMDNJ 1977. Board certified (IM). Solo, partnership, group. Available July 1986.

Prasad Satyanarayana Maturu, M.D., 8863 17th Ave., Brooklyn, NY 11214. Mysore Medical (India) 1977. Board eligible. Group or partnership. Available June 1986.

Brian C. Weiner, M.D., 24 Bowling Green Pl., Staten Island, NY 10314. SUNY-Downstate 1981. Board certified. Group or partnership. Available July 1986.

INTERNAL MEDICINE—Walter M. Bakun, M.D., 199 Casterline Rd., Denville, NJ 07834. St. George's University (Grenada) 1983. Board eligible. Group, partnership, solo. Available July 1986.

Joseph Cohen, M.D., 16 Mountainbrow Village, Corning, NY 14830. New York Medical College 1982. Board eligible. Group. Available July 1986.

Arkady S. Fishman, M.D., 55 Nagle Ave., Apt. 1E, New York, NY 10040. Aktyubinsk Medical Institute 1969. Board certified. House staff, group, nursing home. Available.

Eli R. Goldner, M.D., 2841 W. Rosemont, Chicago, IL 60659. Rome (Italy) 1980. Board eligible. Group, HMO, partnership. Available.

Arpad C. Heinrich, M.D., 360 W. Pleasantview Ave., Hackensack, NJ 07601. Temeschwar (Transylvania) 1980. Board eligible. Solo, partnership, group. Available June 1986.

Serge I. Kaftal, M.D., 125 Gates Ave., Apt. 38, Montclair, NJ 07042. Lodz Medical Academy (Poland) 1978. Solo, group, health center service. Available.

H. Homayouni, M.D., 459 Hamilton Pl., Hackensack, NJ 07601. Tehran (Iran) 1971. Board certified. Partnership. Available.

Rey Chi Lin, M.D., 714 Indian Creek Dr., Wilkes-Barre, PA 18702. China Medical 1977. Board eligible. Group, partnership, solo. Available July 1986.

Gerald B. Miele, M.D., 293 Demott St., Mineola, NY 11501. Rush 1983. Board eligible. Solo or partnership. Available July 1986.

Makkena Ramachandra Rao, M.D., 501 Indian Creek Dr., Wilkes-Barre, PA 18702. Guntur Medical (India) 1974.

Board eligible. Group or partnership. Available July 1986.

Danuta Silber, M.D., 340 West End Ave., NY 10023. UMDNJ (PGY-3), Poland 1979. Board eligible. Group or partnership. Available July 1986.

Marian Lynn Taylor, M.D., 7 Loriann Rd., Warren, NJ 07060. UMDNJ 1983. Board eligible. Group or partnership. Available June 1986.

Dipak Vora, M.D., 509 Indian Creek Dr., Wilkes-Barre, PA 18702. University of Bombay (India). Board eligible. Available July 1986.

OBSTETRICS/GYNECOLOGY—Alan Cohen, M.D., 2335 Charney Rd., University Heights, OH 44118. Dominica 1982. Board eligible. Group, partnership, solo. Available July 1986.

OPHTHALMOLOGY—Kenneth R. Brein, M.D., 55 Beacon St., Pittsburgh, PA 15217. University of Pennsylvania 1982. Board eligible. Group, partnership, solo. Available July 1986.

Andrew R. Gewirtz, M.D., 64 Pond Rd., Apt. G3, Woodbury, NY 11797. Chicago 1978. Board eligible. Board certified (PED). Single or multispecialty group. Available.

PULMONARY—Raymond Mantovani, M.D., 89 Fleet Pl., Mineola, NY 11501. Universidad Centro de Estudios Teenicos (Dominican Republic) 1981. Board eligible. Group, partnership, solo, hospital-based. Available July 1986.

Fred A. Wagshul, M.D., 55 Mill Plain Rd., Unit 16-4, Danbury, CT 06811. Universidad Central del Este 1980. Board eligible. (IM). Group, partnership, hospital-based. Available July 1986.

RADIOLOGY—Jesus S. Santos, Jr., M.D., RD 2, Gibson Rd., Goshen, NY 10924. Santo Tomas (Philippines) 1960. Board certified. Available.

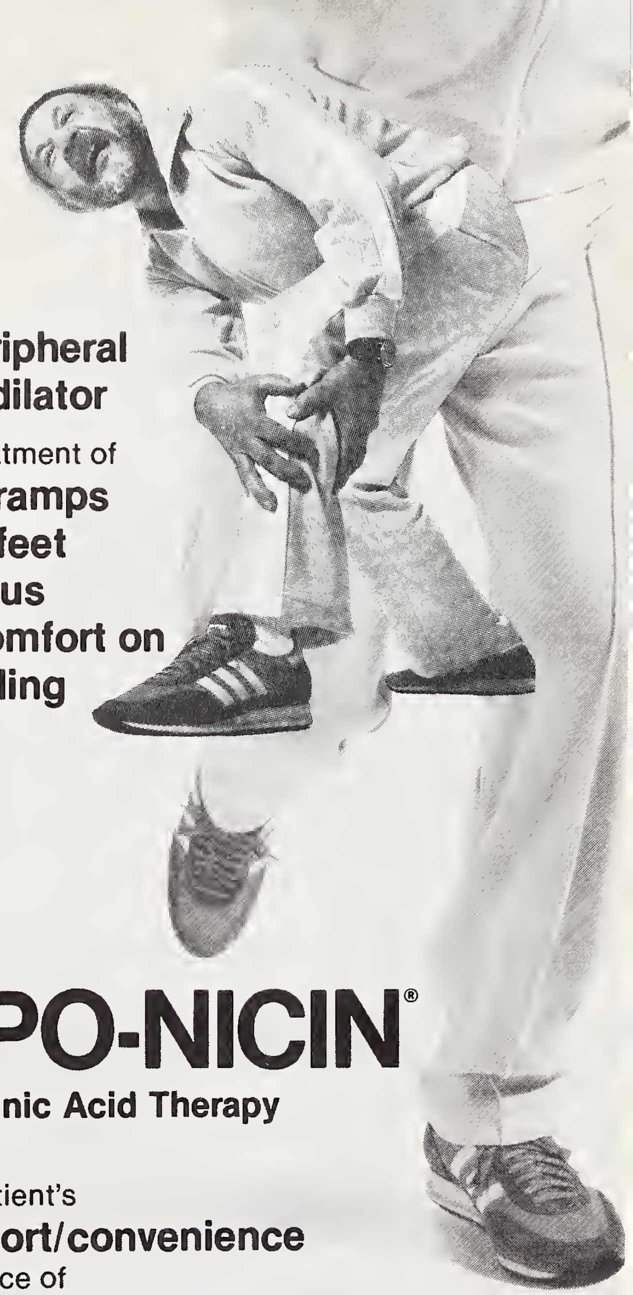
SURGERY, GENERAL—Bernard Henson, M.D., 1275 15th St., Apt. 16-0, Fort Lee, NJ 07024. Guadalajara (Mexico) 1979. Board eligible. Partnership or group. Available July 1986.

Derrick John Martin, M.D., 13-2 Granada Cres., White Plains, NY 10603. West Indies 1977. Board certified. Group, partnership, solo. Available.

Russell H. Zelman, M.D., 309 DeMott St., Mineola, NY 11501. Universidad del Noreste (Mexico) 1980. Board eligible. Group, solo, partnership. Available July 1986.

SURGERY, PLASTIC—Jerrold Zeitel, M.D., 240 E. Montgomery Ave., Apt. 16, Ardmore, PA 19003. Pennsylvania 1980. Board eligible. Group or partnership. Available July 1987.

UROLOGY—Louis C. Galdieri, M.D., 1563 Hampton Knoll Dr., Akron, OH 44313. Guadalajara (Mexico) 1979. Group or partnership. Available August 1986.



A peripheral vasodilator

for treatment of
leg cramps
cold feet
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LIPO-NICIN®/300 mg.

Each time-release capsule contains:

Nicotinic Acid	300 mg.
Ascorbic Acid	150 mg.
Thiamine HCL (B-1)	25 mg.
Riboflavin (B-2)	2 mg.
Pyridoxine HCL (B-6)	10 mg.

in a special base of prolonged therapeutic effect.
DOSE: 1 to 2 tablets daily.
AVAILABLE: Bottles of 100, 500.

Immediate Release
LIPO-NICIN®/250 mg.

Each yellow tablet contains:

Nicotinic Acid	250 mg.
Niacinamide	75 mg.
Ascorbic Acid	150 mg.
Thiamine HCL (B-1)	25 mg.
Riboflavin (B-2)	2 mg.
Pyridoxine HCL (B-6)	10 mg.

DOSE: 1 to 3 tablets daily.
AVAILABLE: Bottles of 100, 500.

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Each blue tablet contains:

Nicotinic Acid	100 mg.
Niacinamide	75 mg.
Ascorbic Acid	150 mg.
Thiamine HCL (B-1)	25 mg.
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Pyridoxine HCL (B-6)	10 mg.

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Indications: For use as a vasodilator in the symptoms of cold feet, leg cramps, dizziness, memory loss or tinnitus when associated with impaired peripheral circulation. Also provides concomitant administration of the listed vitamins. The warm tingling flush which may follow each dose of LIPO-NICIN® 100 mg. or 250 mg. is one of the therapeutic effects that often produce psychological benefits to the patient.

Side Effects: Transient flushing and feeling of warmth seldom require discontinuation of the drug. Transient headache, itching and tingling, skin rash, allergies and gastric disturbance may occur.

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2500 West Sixth Street, Los Angeles, California 90057

REFER TO PDR

Americana Host Farm Resort

HOUSING APPLICATION
220th ANNUAL MEETING
THE MEDICAL SOCIETY OF NEW JERSEY

THURSDAY, MAY 1, TO SUNDAY, MAY 4, 1986

THE FOLLOWING ARE ALL-INCLUSIVE PACKAGE PLANS BASED ON MODIFIED AMERICAN PLAN, INCLUDING DINNER AND BREAKFAST DAILY STARTING WITH DINNER ON THE DAY OF ARRIVAL, THE SATURDAY NIGHT BANQUET, LIVE CABARET SHOWS EACH EVENING FEATURING A DIFFERENT SHOW EVERY NIGHT, STATE SALES TAX, AND ALL GRATUITIES COVERING BELLMEN FOR IN/OUT LUGGAGE SERVICE, CHAMBERMAIDS, AND ALL MEAL SERVICE AS OUTLINED:

FOUR-NIGHT PACKAGE
$435.00 PER PERSON, SINGLE OCCUPANCY
$324.00 PER PERSON, DOUBLE OCCUPANCY
$120.00 PER CHILD SHARING A ROOM WITH PARENTS

THREE-NIGHT PACKAGE
$337.00 PER PERSON, SINGLE OCCUPANCY
$251.00 PER PERSON, DOUBLE OCCUPANCY
$ 90.00 PER CHILD SHARING A ROOM WITH PARENTS

TWO-NIGHT PACKAGE
$238.00 PER PERSON, SINGLE OCCUPANCY
$177.00 PER PERSON, DOUBLE OCCUPANCY
$ 60.00 PER CHILD SHARING A ROOM WITH PARENTS

- Forms received after April 1, will be confirmed on an "if avail basis.
- Fill out only one form for each required.
- DEPOSIT OF $100 PER R MUST ACCOMPANY FORM CONFIRM RESERVATIONS.
- Please make check payable to H FARM. Major credit cards acce at hotel, but not for deposit.
- Hotel confirmation is required check-in.
- Send form and deposit to:
  Host Reservations
  2300 Lincoln Highway East
  Lancaster, PA 17602
  717-299-5500

CHECK TYPE OF ROOM REQUIRED: ☐ SINGLE OCCUPANCY ☐ DOUBLE OCCUPANCY

PLEASE NOTE: Number of persons attending the Inaugural Dinner-Dance honoring President-Elect and M Edward A. Schauer on Saturday, May 3, 1986 ____________

PLEASE PRINT OR TYPE THE FOLLOWING INFORMATION:

NAME: ____________________ NO. of CHILDREN: ________

SHARING ROOM WITH: ____________________ CHILDREN'S AGES: ________

ADDRESS: ____________________

CITY: ____________________

PHONE: __________ STATE: __________ ZIP: __________

CHECK IF OFFICIAL DELEGATE ☐ COUNTY: __________

ARRIVAL DATE __________ DAY __________

DEPARTURE DATE __________ DAY __________

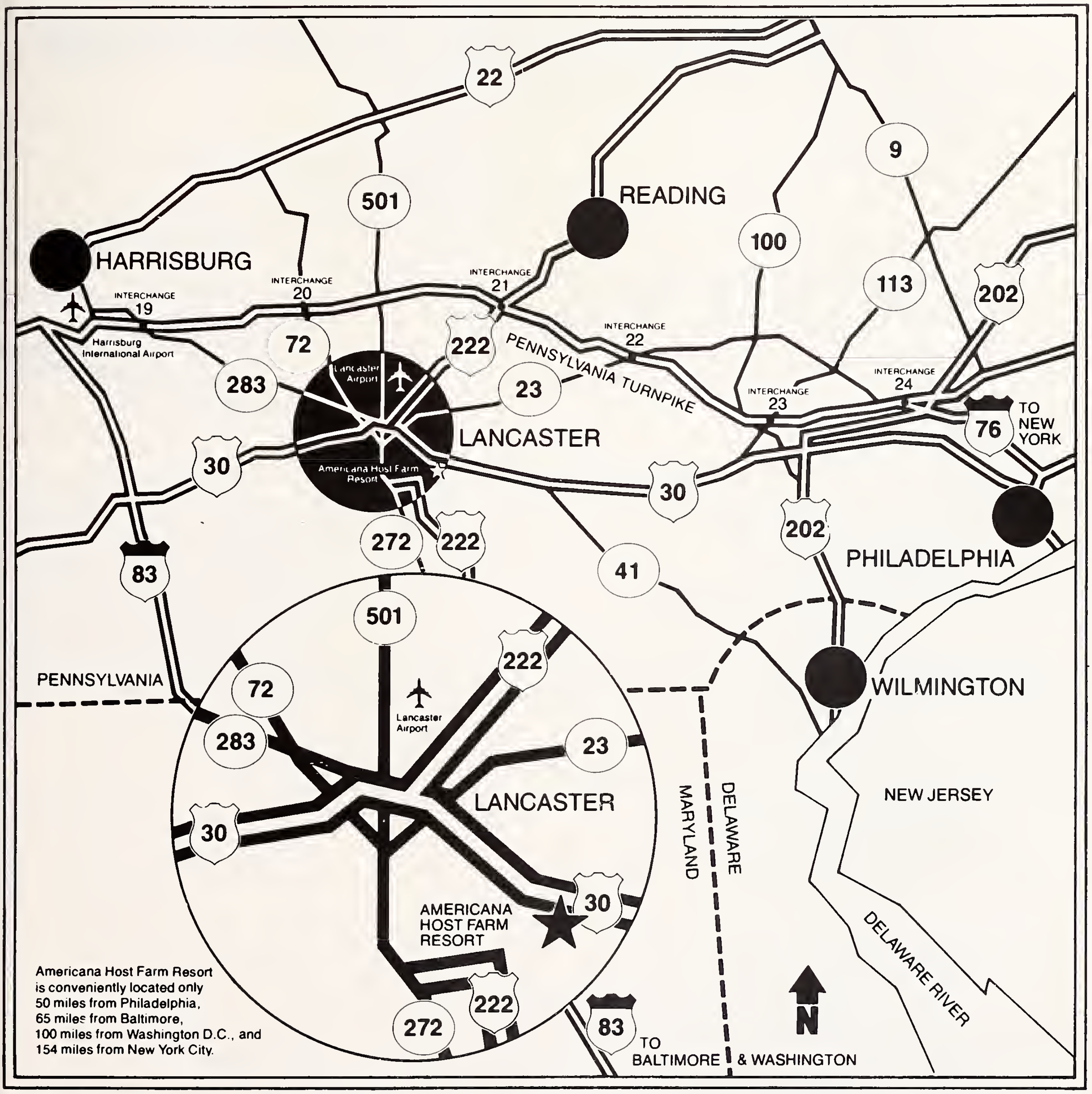
CHECK-IN: AFTER 4 PM
CHECK-OUT: BY 12 NOON

MEDICAL SOCIETY OF NEW JERSEY—220TH ANNUAL MEETING

F-9
C-9

Americana
Host Farm Resort

2300 Lincoln Highway East (Route 30), Lancaster, Pennsylvania 17602 (717) 299-5500



Americana Host Farm Resort is conveniently located only 50 miles from Philadelphia, 65 miles from Baltimore, 100 miles from Washington D.C., and 154 miles from New York City.

FROM NEW YORK

Take New Jersey Turnpike South to Pa. Turnpike. Take Pa. Turnpike West to Interchange No. 21. Travel South on U.S. Rte. 222 to U.S. Route 30. Travel East on U.S. Route 30, approximately 5 miles to Americana Host Farm Resort.

FROM PITTSBURGH & POINTS WEST

Travel East on Pa. Turnpike to Int. 19. Go South on I-283 for 30 miles into Route 30 East for 6 miles to Americana Host Farm Resort.

FROM PHILADELPHIA

Take Pa. Turnpike West to Interchange No. 21. Travel South on U.S. Rte. 222 to U.S. Route 30. Travel East on U.S. Route 30, approximately 5 miles to Americana Host Farm Resort.

FROM BALTIMORE & WASHINGTON

Go North on Interstate Route 83 to York, Pa. Turn East on U.S. Route 30 for 32 miles to Americana Host Farm Resort.

BY AIR

Harrisburg International Airport (just 35 minutes from Host by limousine or car) via TWA and U.S. Air, and Lancaster Airport only minutes away with daily flights via U.S. Air.

BY RAILROAD

Amtrak to Lancaster Station. Taxi and Rent-a-Car Service available.

The Comprehensive Headache Center
at The Germantown Hospital and Medical Center in cooperation with
Temple University School of Medicine, Department of Neurology
Presents

Second Annual—

A HEADACHE SYMPOSIUM

New Advances In Headache Treatment

Moderator: **Elliott A. Schulman, M.D.**
Attending Neurologist and Co-Director of The Comprehensive Headache Center
The Germantown Hospital and Medical Center
Assistant Professor of Neurology, Temple University

Saturday, May 10, 1986 9A.M.–3:30P.M.
at The Germantown Hospital and Medical Center

One Penn Boulevard, Philadelphia, Pa.
(adjacent to LaSalle University at the intersection
of Wister Street, Chew and Olney Avenues)

Featured Speakers and Topics:

Introduction and Welcoming Remarks

Wallace McCune, M.D.
Professor of Medicine
Temple University School of Medicine
Chairman, Department of Medicine
The Germantown Hospital and Medical Center

New Concepts in the Treatment of Migraine

Stephen D. Silberstein, M.D.
Chief, Neurology Section & Co-Director
The Comprehensive Headache Center
Associate Professor of Neurology, Temple University

Analgesic Rebound and Chronic Muscle Contraction Headache

Alan Rapoport, M.D.
Co-Director, The New England
Center for Headache, Cos Cob, Connecticut

Cluster Headache

Lee Kudrow, M.D.
Director, California Medical Clinic
for Headache
Encino, California

Post Traumatic Headaches

Joel Saper, M.D.
Director, Michigan Headache and
Neurological Institute, P.C.
Clinical Associate Professor of
Medicine (Neurology)
University of Michigan
Ann Arbor, Michigan

Additional Participants will include:

Ronald S. Kaiser, Ph.D., Clinical Psychologist and Co-Director, The Comprehensive Headache Center; Assistant Professor of Psychology, Hahnemann University.

Gregory J. Tramuta, M.D., Psychiatrist and Co-Director, The Comprehensive Headache Center; Clinical Assistant Professor of Psychiatry, Temple University.

Joseph P. Primavera III, Staff Psychologist and Co-Director, The Comprehensive Headache Center; Vocational Services Director of The Rehabilitation Diagnostic Center, The Germantown Hospital and Medical Center.

A roundtable discussion will include controversial topics in headache management and difficult cases.

* Plan to bring your difficult cases for discussion.

Credit:

4.5 credit hours in Category I credit
provided it is completed as designed.
A.A.F.P. prescribed credits.
AOA credit.

Registration Fee:

$15.00 includes coffee breaks, lunch and registration material.

Please make checks payable to
The Germantown Hospital and Medical Center
and send by May 1, 1986 to:

The Comprehensive Headache Center
The Germantown Hospital and Medical Center
One Penn Boulevard Philadelphia, PA 19144

CME CALENDAR

The following is a list of continuing medical education courses for the next two months. Contact the sponsoring organization for further information.

This list is compiled through the cooperation of the Committee on Medical Education of the Medical Society of New Jersey, The Academy of Medicine of New Jersey, the New Jersey Chapter of the American Academy of Family Physicians, and the Office of Continuing Medical Education of the UMDNJ. For information on accreditation, please contact the sponsoring organization(s), indicated by italics—last line of each item.

ANESTHESIOLOGY

May
5 **Anesthesia Lectures**
12 7 A.M.—Medical Science Bldg.,
19 E-545, NJ Medical School, Newark
(UMDNJ)

5 **Anesthesia Lectures**
12 7:45 A.M.—Auditorium, 3rd Fl.,
19 Newark Beth Israel Medical Center
(UMDNJ)

6 **Anesthesia Lectures**
13 4 P.M.—Medical Science Bldg.,
20 E-545, NJ Medical School, Newark
27 *(UMDNJ)*

28 **Anesthesia Grand Rounds**
4-5 P.M.—University Hospital,
H-350, Newark
(UMDNJ)

June
2 **Anesthesia Lectures**
9 7:45 A.M.—Auditorium, 3rd Fl,
16 Newark Beth Israel Medical Center
23 *(UMDNJ)*
30

2 **Anesthesia Lectures**
9 7 A.M.—Medical Science Bldg.,
16 E-545, NJ Medical School, Newark
23 *(UMDNJ)*
30

3 **Anesthesia Lectures**
10 4 P.M.—Medical Science Bldg.,
17 E-545, NJ Medical School, Newark
24 *(UMDNJ)*

25 **Anesthesia Grand Rounds**
4-5 P.M.—University Hospital,
H-350, Newark
(UMDNJ)

CARDIOLOGY

May
8 **Deep Vein Thrombosis and Pulmonary Embolism**
11 A.M.-12 noon—St. Joseph's Hospital and Medical Center, Paterson
(St. Joseph's Hospital and Medical Center)

30 **Cardiothoracic Journal Club**
7:30 P.M.—13 Ridgeway Drive, West Orange
(UMDNJ)

June
27 **Cardiothoracic Journal Club**
7:30 P.M.—13 Ridgeway Drive, West Orange
(UMDNJ)

MEDICINE

May
1 **Case Seminars**
15 8-10 P.M.—312 Harding Drive, South Orange
(Advanced Psychiatric Study Group and AMNJ)

3 **Life-Threatening Crisis in the Emergency and Intensive Care Units**
9 A.M.-3 P.M.—Rutgers Medical School, New Brunswick
(UMDNJ and AMNJ)

5 **Blood Banking Conferences**
12 11 A.M.—Medical Science Bldg.,
19 C-128, NJ Medical School, Newark
27 *(UMDNJ)*

5 **A Delayed Reaction to Rape**
8:15-10:15 P.M.—543 Park Street, Upper Montclair
(Essex Psychiatric Seminar and AMNJ)

7 **Grand Rounds in Psychiatry**
10:30 A.M.-12:30 P.M.—Medical Science Bldg., B-610, NJ Medical School, Newark
(UMDNJ)

8 **Deep Vein Thrombosis and Pulmonary Embolism**
11 A.M.-12 noon—St. Joseph's Hospital and Medical Center
(St. Joseph's Hospital and Medical Center)

13 **Hypertension in the Elderly**
9-10 A.M.—Holy Name Hospital, Teaneck
(Holy Name Hospital and AMNJ)

21 **Psychiatry Lecture**
10:30 A.M.-12:30 P.M.—Medical Science Bldg., B-610, NJ Medical School, Newark
(UMDNJ)

21 **Psychiatric Lecture**
1-3 P.M.—Essex County Hospital Center, Cedar Grove
(UMDNJ)

24 **Behavioral Medicine**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

29 **Treatment of Herpes Disease in Immunocompromised Patients**
11 A.M.-12 noon—St. Joseph's Hospital and Medical Center, Paterson
(St. Joseph's Hospital and Medical Center)

June
Mini-Residency in Proctology and Disease of the Colon
One week commitment—Contact Overlook Hospital, Summit
(Overlook Hospital and AMNJ)

2 **Blood Banking Conference**
9 11 A.M.—Medical Science Bldg.,
16 C-128, NJ Medical School, Newark
23 *(UMDNJ)*
24
30

3 **Ototoxic Drugs**
7-8 P.M.—West Hudson Hospital, Kearny
(West Hudson Hospital and AMNJ)

3 **Pulmonary Conference**
10 8-9 A.M.—University Hospital,
17 Newark
24 *(UMDNJ)*

4 **Medical Lecture Series**
11 10:30 A.M.-12 noon—Christ Hospital, Jersey City
(Christ Hospital and AMNJ)

4 **Rehabilitation Medicine**
11 4:15-5:15 P.M.—Kessler Institute,
18 West Orange
25 *(UMDNJ)*

5 **Trauma Conference**
12 7:45-8:45 A.M.—University Hospital,
19 H-349, Newark
26 *(UMDNJ)*

5 **Digestive Disease Case Report**
12 8-9 A.M.—Medical Science Bldg.,
19 H-249, NJ Medical School, Newark
26 *(UMDNJ)*

5 **Digestive Disease Research and**
12 **Literature Review Conference**
12 9-10 A.M.—Medical Science Bldg.,
26 H-249, NJ Medical School, Newark
(UMDNJ)

5 **Allergy/Immunology Lecture**
12 11 A.M.-12 noon—United Hospital,
19 Newark
26 *(UMDNJ)*

6 **Cancer Research Colloquium**
13 12 noon-1 P.M.—Medical Science
20 Bldg., G-506, NJ Medical School,
27 Newark
(UMDNJ)

6 **Dept. of Medicine Grand Rounds**
13 12 noon-1 P.M.—Medical Science
20 Bldg., B-610, NJ Medical School,
27 Newark
(UMDNJ)

6 **Urology Basic Surgery Conference**
13 2-3 P.M.—Medical Science Bldg.,
20 C-600, NJ Medical School, Newark
27 *(UMDNJ)*

6 **Urology Grand Rounds**
13 3-5 P.M.—Medical Science Bldg.,
20 C-600, NJ Medical School, Newark
27 *(UMDNJ)*

10 **An Overview of Cancer Pain**
17 **Drug-Induced Hepatic Toxicity**
9-10 A.M.—Holy Name Hospital, Teaneck
(Holy Name Hospital and AMNJ)

June 9-13, 1986

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WEDNESDAY, MAY 7, 1986

REDUCTION OF INFARCT SIZE: CAN IT BE DONE?

MODERATOR: BERNARD L. SEGAL, M.D.

3:00	Roundtable Discussion and Debate:	
	Streptokinase and Coronary Angioplasty are Appropriate	*Charles E. Bemis, M.D.*
	Conservative Management is Appropriate	*Sheldon R. Bender, M.D.*
5:00	Questions and Answers	*The Audience*

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13 **Current Clinical Concepts in Rheumatology/Immunology**
12:15-1:15 P.M.—Kennedy Memorial Hospital, Stratford
(UMDNJ)

16 **IVP Conference**
5-9 P.M.—East Orange VA Hospital, Rm. 142
(UMDNJ)

18 **Interhospital Rheumatology Rounds**
4:30-6:30 P.M.—University Hospital, I-355, Newark
(UMDNJ)

19 **Managing Hepatitis**
5-6:30 P.M.—Somerset Medical Center, Somerville
(Somerset Medical Center and AMNJ)

NEUROLOGY/PSYCHIATRY

May

1 **Case Seminars**
15 8-10 P.M.—312 Harding Drive, South Orange
(Advanced Psychiatric Study Group and AMNJ)

5 **A Delayed Reaction to Rape**
8:15-10:15 P.M.—543 Park Street, Upper Montclair
(Essex Psychiatric Seminar and AMNJ)

7 **Grand Rounds in Psychiatry**
10:30 A.M.-12:30 P.M.—Medical Science Bldg., B-610, NJ Medical School, Newark
(UMDNJ)

21 **Psychiatry Lecture**
10:30 A.M.-12:30 P.M.—Medical Science Bldg., B-610, NJ Medical School, Newark
(UMDNJ)

21 **Psychiatric Lecture**
1-3 P.M.—Essex County Hospital Center, Cedar Grove
(UMDNJ)

24 **Behavioral Medicine**
8 A.M.—Newcomb Hospital, Vineland
(Newcomb Hospital)

June

2 **Pervasive Developmental Disorder Grown Up**
8:15-10:15 P.M.—1046 South Orange Ave., Short Hills
(Essex Psychiatric Seminar and AMNJ)

4 **Grand Rounds in Psychiatry**
10:30 A.M.-12:30 P.M.—Medical Science Bldg., 610, NJ Medical School, Newark
(UMDNJ)

5 **Case Seminars**
19 8-10 P.M.—312 Harding Drive, South Orange
(Advanced Psychiatric Study Group and AMNJ)

7 **The Adolescent Family: Teenagers and Their Children**
9 A.M.-4 P.M.—NJ Medical School, Newark
(UMDNJ and AMNJ)

18 **Psychiatric Lecture**
10:30 A.M.-12:30 P.M.—Medical Science Bldg., B-610, NJ Medical School, Newark
(UMDNJ)

18 **Psychiatry Lecture**
1-3 P.M.—Essex County Hospital Center, Cedar Grove
(UMDNJ)

OBSTETRICS/GYNECOLOGY

May

6 **Pathology/Ob/Gyn Conference**
13 5:15-6:15 P.M.—Medical Science
20 Bldg., C-579, NJ Medical School,
27 Newark
(UMDNJ)

PATHOLOGY

May

1 **Pathology/Radiology/Orthopedics**
8 **Conference**
15 8-9 A.M.—University Hospital,
22 Newark
29 *(UMDNJ)*

1 **Pathology Experimental Seminar**
8 12 noon-1 P.M.—Medical Science
15 Bldg., C-555, NJ Medical School,
22 Newark
29 *(UMDNJ)*

6 **Anatomic Pathology**
13 5:15 P.M.—Medical Science Bldg.,
20 C-555, NJ Medical School, Newark
27 *(UMDNJ)*

6 **Pathology/Ob/Gyn Conference**
13 5:15-6:15 P.M.—Medical Science
20 Bldg., C-579, NJ Medical School,
27 Newark
(UMDNJ)

7 **Neuropathology Conference**
21 10:30-12:30 P.M.—Medical Science Bldg., C-555, NJ Medical School, Newark
(UMDNJ)

7 **Pathology Experimental Seminar**
21 12 noon-1 P.M.—Medical Science Bldg., C-555, NJ Medical School, Newark
(UMDNJ)

7 **Pathology Grand Rounds**
14 1-5 P.M.—Medical Science Bldg.,
21 C-555, NJ Medical School, Newark
28 *(UMDNJ)*

June

3 **Anatomic Pathology**
10 5:15 P.M.—Medical Science Bldg.,
17 C-555, NJ Medical School, Newark
24 *(UMDNJ)*

4 **Neuropathology Conference**
18 10:30 A.M.-12:30 P.M.—Medical Science Bldg., C-555, NJ Medical School, Newark
(UMDNJ)

4 **Pathology Experimental Seminar**
18 12 noon-1 P.M.—Medical Science Bldg., C-555, NJ Medical School, Newark
(UMDNJ)

4 **Pathology Grand Rounds**
11 1-5 P.M.—Medical Science Bldg.,
18 C-555, NJ Medical School, Newark
25 *(UMDNJ)*

5 **Pathology/Radiology/Orthopedics**
12 **Conferences**
19 8-9 A.M.—University Hospital,
26 Newark
(UMDNJ)

5 **Pathology Experimental Seminar**
12 12 noon-1 P.M.—Medical Science
19 Bldg., C-555, NJ Medical School,
26 Newark
(UMDNJ)

28 **Soft Tissue Workshop**
29 8 A.M.-4:30 P.M.—NJ Medical School, Newark
(UMDNJ and ANNJ)

PEDIATRICS

May

2 **Advances in Pediatrics**
9 9:30-10:30 A.M.—Medical Science
16 Bldg., B-610, NJ Medical School,
23 Newark
30 *(UMDNJ)*

13 **Common Pediatric Ophthalmological Problems**
9:30 A.M.-10:30 A.M.—Elizabeth General Medical Center
(Newark Beth Israel Medical Center and AMNJ)

28 **Pediatric Update—1986**
9:10 A.M.-4:30 P.M.—The Meadowlands, East Rutherford
(Children's Hospital of NJ and AMNJ)

June

6 **Advances in Pediatrics**
13 9:30-10:30 A.M.—Medical Science
20 Bldg., B-610, NJ Medical School,
27 Newark
(UMDNJ)

RADIOLOGY

May

1 **Pathology/Radiology/Orthopedics**
8 **Conference**
15 8-9 A.M.—University Hospital,
22 Newark
29 *(UMDNJ)*

7 **Radiology Grand Rounds**
21 2-4 P.M.—University Hospital, H-346, Newark
(UMDNJ)

June

4 **Radiology Grand Rounds**
18 2-4 P.M.—University Hospital, H-346, Newark
(UMDNJ)

5 **Pathology/Radiology/Orthopedics**
12 **Conference**
19 8-9 A.M.—University Hospital,
26 Newark
(UMDNJ)

SURGERY

May

3 **Surgical Treatment of Cardiothoracic Diseases**
10 A.M.-12 noon—Medical Science Bldg., G-506, NJ Medical School, Newark
(UMDNJ)

3 **Surgical Morbidity and Mortality**
10 **Conference**
17 8:30-9:30 A.M.—Medical Science
24 Bldg., G-506, Newark
(UMDNJ)

5 **Tumor Conference**
12 8-9 A.M.—University Hospital,
19 Newark
(UMDNJ)

5 **Surgical Grand Rounds and**
12 **Lecture Series**
19 4:30-6:30 P.M.—Medical Science Bldg., B-610, NJ Medical School, Newark
(UMDNJ)

6 **Surgical Grand Rounds**
7-9 A.M.—Hackensack Medical Center

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June 21, 1986

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10 **Microsurgery (Neurovascular—Tendon)**
8 A.M.—Newcomb Hospital Vineland
(Newcomb Hospital)

27 **Pediatric Surgery-Ambiguous Genitalia**
8-10 P.M.—Englewood Club, 115 E. Palisade Ave., Englewood
(Englewood Surgical Society and AMNJ)

June

2 **Tumor Conference**
9 8-9 A.M.—University Hospital,
16 H-349, Newark
23 *(UMDNJ)*
30

7 **Surgical Treatment of Cardiothoracic Diseases**
10 A.M.-12 noon—Medical Science Bldg., G-506, NJ Medical School, Newark
(UMDNJ)

7 **Surgical Morbidity and Mortality**
14 **Conference**
21 8:30-9:30 A.M.—Medical Science
28 Bldg., G-506, NJ Medical School,
Newark
(UMDNJ)

SURGICAL SPECIALTIES

May

Pathology/Radiology Orthopedics
1 **Conference**
8 8-9 A.M.—University Hospital.
15 Newark
22 *(UMDNJ)*

5 **Orthopedic Grand Rounds**
12 6-8 P.M.—Medical Science Bldg.,
19 B-556, NJ Medical School, Newark
(UMDNJ)

15 **Neurosurgery Lecture**
4-6 P.M.—University Hospital, E-343, Newark
(UMDNJ and AMNJ)

June

2 **Orthopedic Grand Rounds**
9 6-8 P.M.—Medical Science Bldg.,
16 B-556, NJ Medical School, Newark
23 *(UMDNJ)*
30

5 **Pathology/Radiology/Orthopedics**
12 **Conference**
19 8-9 A.M.—University Hospital,
26 Newark
(UMDNJ)

12 **Neurosurgery Lecture**
4-6 P.M.—University Hospital, E-343, Newark
(UMDNJ)

18 **Grand Rounds in Ophthalmology**
7-9 P.M.—United Hospitals Annex Bldg., Newark
(UMDNJ)

MISCELLANEOUS

May

14 **Office Management of Common Medical Problems**
12 noon-4 P.M.—Kennedy Memorial Hospital, Stratford
(UMDNJ)

June

11 **Office Management of Common Medical Problems**
12 noon-4 P.M.—Kennedy Memorial Hospital, Stratford
(UMDNJ)

220th MSNJ ANNUAL MEETING

PROPOSED DAILY SCHEDULE

May 1-4, 1986
Americana Host Farm Resort
Lancaster, Pennsylvania

Wednesday, April 30, 1986
3:30 P.M. —Board of Trustees' meeting

Thursday, May 1, 1986
7:30 A.M. —Registration open
9:00 A.M. —Message Center and Exhibits open
2:00 P.M. —House of Delegates
4:00 P.M. —Reference Committees

Friday, May 2, 1986
8:00 A.M. —Registration, Message Center, and Exhibits open
9:00 A.M. —House of Delegates (election)
12:00 NOON —Golden Merit Award Ceremony followed by Reception
2:30 P.M. —Reference Committees
5:00 P.M. —JEMPAC Political Forum
5:45 P.M. —JEMPAC Wine & Cheese Reception

Saturday, May 3, 1986
8:00 A.M. —Registration, Message Center, and Exhibits open
8:30 A.M. —House of Delegates
1:30 P.M. —House of Delegates
6:30 P.M. —Inaugural Reception followed by Inaugural Dinner

Sunday, May 4, 1986
8:00 A.M. —Registration and Message Center open
8:30 A.M. —General Session—"Corporate Invasion in the Practice of Medicine"
1:00 P.M. —Board of Trustees' meeting

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LETTERS TO THE EDITOR

Smoking and Hypnosis; Viral Diagnostic Laboratory

Smoking and Hypnosis

January 6, 1986

Dear Dr. Krosnick:

Enough talk about the dangers of smoking. Let's get our patients off it. When you devote 45 minutes to a patient and help him to quit smoking two packs of cigarettes a day, you can be adding five to ten years to his life, as well as several thousand dollars—not a bad investment.

When patients come to me to stop smoking, I use formal hypnosis. Hypnosis, however, need not be formal nor obvious.

The next time a smoker comes into your office, grab his attention. Whatever he came in for, listen to his chest and ask him to cough, breath deeply, cough again, and listen over his heart. Then look him in the eye and solemnly ask, "How much have you been smoking lately?" "Have you been spitting up any blood recently?" When he replies, "Why do you ask," don't answer but go back and listen to his chest again.

Tell him to go to the radiology service and get a chest x-ray now. Then he is to go home and wait for your call. You now have gained his attention and cooperation (the basic elements of hypnosis). Let him sweat at home for a while (giving him time to ask himself why he hadn't quit smoking when he had the chance). Have your secretary call the patient. When he gets on the line have him wait. Instead of getting on, have your nurse pick up, then (after a long pause) you pick up. Say something like, "Harry, I just got off the phone with the radiologist."

"Well Doc, what is it—is something wrong?"

"Well Harry, there's good news and bad news."

"What is it?"

"The good news is that the radiologist doesn't see any signs of emphysema or cancer, yet, in today's x-ray."

"What do you mean, yet, and what's the bad news?"

"Well there's some early signs of parenchymal scarring."

"What does all that mean?"

"No cause for alarm, Harry, but try to understand."

"Understand what?"

"Sometimes people who smoke and have parenchymal scarring at your age, go on to get lung cancer or emphysema."

"What are you saying?"

"If I were you, Harry, I'd go take every cigarette that I own, break them apart, throw them out, kiss the wife and kids, and thank God that I got off those dried up leaves before the damage was irreparable."

"You mean if I don't smoke any more I'll be okay?"

"In all likelihood, yes; but remember Harry, you now are and will always be a nicotine addict. You will never be able to have "just one." When tempted to have a cigarette, take a deep breath in, blow it out, and remember that beautiful family of yours. Keep me posted, and let me know if you need any help."

There is a power in the sight of a white coat and stethoscope, the smell of tincture and benzoin, and the touch and sound of a caring physician. Use that power … and "May the Force be with you."

(signed) Robert G. Jarmon, M.D.

Viral Diagnostic Laboratory

January 31, 1986

Dear Dr. Krosnick:

Last year, 1985, marked the second full year of operation of the Rutgers Medical School Viral Diagnostic Laboratory. A total of 1,635 specimens were submitted for viral identification. This is double the number submitted in 1984. Three hundred twenty viruses were identified, for a 20 percent isolation rate. Again, herpes simplex virus was the top virus isolated with rotavirus second and enterovirus third (Table). To my knowledge, the 44 enteroviruses isolated manifest a rather prolonged and intensive epidemic more significant than I personally ever have experienced before. The enterovirus strains isolated include: Coxsackie B1, B4; Echo 4, 7, 9, and 11.

TABLE

Rutgers Medical School Viral Diagnostic Laboratory

Total specimens submitted	1,635
Viruses identified	320
Percent positive	(20%)
Herpes simplex virus	150
Rotavirus	81
Enterovirus	44
Cytomegalovirus	42
Herpes varicella zoster	1
Adenovirus	1
Respiratory syncytial virus	1

Rapid identification techniques for herpes simplex virus, rotavirus, cytomegalovirus, herpes varicella zoster virus, adenovirus, and respiratory syncytial virus (RSV) are in place and seem to be working. We are looking for our first influenza virus isolate of the 1986 season, and already have had a number of RSV isolates for the 1986 season. We continue to hope that our efforts will allow the physicians of central New Jersey a resource for the most up-to-date viral diagnosis. This becomes even more important with the release of ribovarin, a specific antiviral agent, for use in respiratory syncytial virus infections and influenza virus infections that may be a threat to compromised infants and young children.

(signed) Lawrence D. Frenkel, M.D.,
Director, Rutgers Medical School
Viral Diagnostic Laboratory

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Advances in Myocardiology; Computed Body Tomography; Current Surgical Diagnosis and Treatment; Hematology, the Blossoming of a Science; Immunology in Obstetrics and Gynecology; 1985 Year Book of Diagnostic Radiology

Advances in Myocardiology, Volume 6

N.S. Dhalla and D.J. Hearse (eds). New York, NY, Plenum Medical Book Company, 1985. Pp. 669. Illustrated. ($79.50).

This book is a collection of papers presented at the 1983 proceedings of the World Congress of the International Society for Heart Research. It is a formidable presentation of advances in the physiology, pharmacology, and metabolism of the normal and abnormal heart. Most of the work is very fundamental research, conducted by respected investigators in a variety of areas including calcium, phospholipid, and nucleotide metabolism, as well as ischemic-reperfusion injury, the electrogenesis of arrhythmias, and cellular mechanisms in contractile failure.

Basic contributions to the study of arrhythmias include evaluation of the intrinsic innervation of the atrioventricular node, action potentials of human atria, the role of the cholinergic system in arrhythmogenesis, the electrophysiologic properties of ischemic myocardium, beta-adrenoceptor blockade, and the development of a model for reperfusion-induced ventricular fibrillation.

Many aspects of the ischemic myocardium are described including the ultrastructure of the sarcoplasmic reticulum, and the effects of adenosine, AMP, prostaglandins, calcium antagonists, glucose, and the pyruvate dehydrogenase complex on anaerobic metabolism.

At least six reports deal with inotropy and contractile failure in terms of calcium influx and agonism, potassium balance and extracellular accumulation, hydrogen ion concentration, isoproterenol infusion, and the mechanism of action of amrinone. There are several additional investigations related to various perturbations and the sodium pump.

Lesser contributions concern alterations in infarct size employing calcium antagonists, anesthetics and flurbiprofen; the role of prostaglandins and calcium antagonists in the reperfusion phenomenon; the evaluation of bioenergetic changes by magnetic resonance; and a fascinating series on etiologic considerations in cardiomyopathy.

It is regrettable that this comprehensive tome will appeal to a relatively small audience. The uninitiated, the casual reader, and the busy clinician will surely eschew its contents. Yet, as before, today's basic research will provide the scientific background for tomorrow's practice of cardiology.

Edwin L. Rothfeld, M.D.

Computed Body Tomography

Joseph K.T. Lee, Stuart S. Sagel, Robert J. Stanley (eds). New York, NY, Raven Press, 1982. Pp. 602. ($89.50)

This concise textbook provides an excellent, highly clinically oriented review of extracranial computed tomography (CT).

After a brief chapter on the history of CT, the editors provide an extensive review of practical techniques of scanning.

Included is a general approach to each area of the body along with scan slice and contrast parameters. It should be noted that some centers rely more on the use of intravenous contrast than do these editors. The neck, an area more commonly discussed in neuroradiology texts, received some attention in the chapter on the larynx. The thorax and abdomen are reviewed in detail with the use of high-quality diagrams. A chapter on the spine provides a brief review of acquired and congenital disease. The chapter on the musculoskeletal system touches briefly on the new application of CT.

While the book holds 602 pages, many of them are filled with moderate quality scans and thus relatively short. I found the text very well written; it is highly recommended for the physician who desires a firm basis for body scan interpretation.

Neil B. Horner, M.D.

Current Surgical Diagnosis and Treatment, 7th Edition

Lawrence W. Way, M.D., (ed). Los Altos, CA, Lange Medical Publications, 1985. Pp. 1,226. Illustrated. ($31.50)

The most recent update of the Lange Series surgical text continues its previous format and fulfills a definite need in the surgical library. The text is characterized by brevity and intensive editorial review. Dr. Way, in the tradition of Dr. Dunphy with whom he previously coauthored this text, has continued to strongly emphasize clinical diagnosis, in such chapters as the "Acute Abdomen" and the peritoneal cavity. His personal authorship of the chapters pertaining to gastrointestinal disease are current, and constitute a well-written review.

The chapter on physical examination is excellent for beginning students of surgery. The chapters on trauma have been updated and provide a concise state-of-the-art review. The chapter on breast diseases incorporates the radical changes in surgical approach which have occurred in the past several years.

Chapters on medical problems in the surgical patient, wounds, inflammation, and the subspecialty chapters are well written and appropriate for inclusion in a surgical text. The chapters on intensive care, amputation, and anesthesia cover areas which are of common interest to most surgical personnel and provide an integrated overview. The chapters on legal aspects of surgical care and radiotherapy appear to be too detailed for a basic surgical text.

The strength of this text rests in

brevity and editorial practice. The author and contributors are located predominantly in a single institution. Revisions are updated every two years. The redundancy so common to most texts is actively addressed by the editor with tight cross-referencing. This is a most refreshing feature of this volume. In short, this is an excellent review text which is highlighted by its currency and the attention to precise editorial detail.

Frank T. Padberg, Jr., M.D.

Hematology, The Blossoming of a Science

Maxwell M. Wintrobe. Philadelphia, PA, Lea & Febiger, 1985. Pp. 563. Illustrated. ($60)

This is a fascinating, well-written, hardcover book and, in these times, well worth the price. It is divided into three principal parts: Part One "From Antiquity to the 1920s," is comprised of 66 pages. Part Two, also 66 pages, is entitled "A Hematologic Odyssey." In this part is included an autobiography of Wintrobe. Part Three is "The Golden Age of Hematology" and contains 360 pages.

The illustrations are photographs of the many scientists who have contributed so much to modern hematology. In a sense, this book is not only about current hematology but also a series of minibiographies of the men and women who are responsible for the astonishing advances in this field.

There is an epilogue in which Dr. Wintrobe states, "My purpose in writing this book has been to record the experiences and contributions of the hundreds of inquisitive and imaginative individuals whose efforts have led to the phenomenal developments in hematology that we witness today." The book has fulfilled that purpose completely. He finally states that one "can compare research with the climbing of mountains. There always are further peaks and we must persist in attempting to reach them." There are many and there will be more men and women who have the qualities that make new and greater achievements possible.

This book is so complete it is difficult to describe all that is here. It includes morphologic hematology, news concerning the clotting factors, all the various red cell dyscrasias, the lymphomas, and a final chapter on cancer chemotherapy. Wintrobe lists 186 references. There is an index of 14 pages in double columns.

I strongly recommend this book to all physicians with any interest in hematology, to medical oncology residents, and to residents in pathology. In fact, I believe all physicians would profit from reading this book by a man whom I believe to be the most distinguished hematologist of this century.

Hugh F. Luddecke, M.D.

Immunology in Obstetrics and Gynecology

James R. Scott and Neal S. Rote. Norwalk, CT, Appleton-Century-Crofts, 1985. Pp. 286

Whether we like it or not, immunology is a part of our professional practice. This is particularly true in obstetrics and gynecology. The mystery of why a mother does not reject her fetus in utero, autoimmune infertility, immunologic diseases in pregnancy, acquired immunodeficiency syndrome, and toxemia of pregnancy all have immunologic components. In addition to covering the above subjects, the authors of this small text wisely include a nine-page glossary. The field is so new and changing so quickly that terminology constantly must be defined and refined.

This book is a good introduction to the subject. It is not an "easy read" but the knowledge must be acquired if the physician is to keep up in this expanding field. It is recommended for medical students, residents, and practitioners of the art and science of obstetrics and gynecology.

Gerard F. Hansen, M.D.

1985 Year Book of Diagnostic Radiology

David G. Bragg, M.D., (ed). Chicago IL, 1985. Pp. 600. Illustrated ($44.95)

The *Year Books* of radiology series were introduced to the medical profession in 1932. The current 1985 *Year Book of Diagnostic Radiology* follows the longstanding successful format of this series by providing succinct and scholarly abstracts o scientific articles published during the preceding year from many sources, followed by concise and informative commentary by the renowned editorial staff assembled by the senior editor of the series, David G. Bragg, M.D.

The book is divided into seven sections, including articles in the following regions of interest: neuroradiology, cardiovascular and interventional radiology, thorax, abdomen, skeletal system, pediatric radiology, and radiation physics. The material contained in these sections was obtained by a review of more than 6,862 articles from 550 journals reflecting a spectrum of the world literature. From these sources, the editorial staff selected 346 articles from 79 journals which should be of special interest to the practicing radiologist. The abstracts are concise and cogent. The editorial comments which follow many of the articles add further insight into the subject matter of the original article, and often add supporting or contrary opinions from references to other original articles previously published in the literature.

This series provides a rapid and conveniently organized method for the busy radiologist to survey the relevant literature compiled from many sources during the preceding year, and nicely augments the more conventional "reprint file" maintained by many specialists. In my opinion, the *Year Book of Diagnostic Radiology* should be a permanent part of the library of every radiologist.

Edwin S. Wilson, M.D.

OBITUARIES

Drs. Dolganos; Epstein; Kloby; Legato; Marquis; Mauer; McCallum; McGeary; Mickewich; Newbury; Pollack; Weiler

Dr. Moses Dolganos

At the advanced age of 86, Moses Dolganos, M.D., a member of our Hudson County component, died on December 20, 1985, at Christ Hospital, Jersey City. Born in New York City, Dr. Dolganos received his medical degree from Ohio State University College of Medicine in 1924. An internist, Dr. Dolganos maintained a practice in Jersey City for more than 50 years. He was a member of the American Medical Association, and for most of his career had been affiliated with Christ Hospital. In 1974, Dr. Dolganos was a recipient of MSNJ's Golden Merit Award, honoring his 50 years as a physician.

Dr. Joshua Epstein

Joshua Epstein, M.D., an ophthalmologist with offices in New York City, died on December 17, 1985, at his home in Union City. Born in Russia in 1905, Dr. Epstein emigrated to the United States at an early age, was graduated from Harvard University at the age of 19, and had to wait a year to enter Tufts University School of Medicine, Boston. He received his medical degree from Tufts in 1926, began his medical practice in Salem, Massachusetts, and within a few years established an office in New York City where he practiced for more than 40 years.

Dr. John J. Kloby

A retired general practitioner, John Joseph Kloby, M.D., died on December 27, 1985, at the Medi-Center of America, Lakewood, at the age of 76. Born in Newark, Dr. Kloby received his medical degree from Georgetown University School of Medicine, Washington, D.C., in 1936. He was a member of our Ocean County component, and of the American Medical Association. Dr. Kloby served the community of Seaside Heights for many years, and was affiliated with Kimball Medical Center, Lakewood, and Community Memorial Hospital, Toms River. During World War II, Dr. Kloby served in the medical corps for three years, emerging with the rank of captain.

Dr. Samuel F. Legato

A surgeon who had practiced in Cliffside Park for over 50 years, Samuel Frank Legato, M.D., died on November 27, 1985, at the age of 78. Dr. Legato was graduated from Bellevue Hospital Medical College, New York, in 1931. He was a member of our Bergen County component, and of the American Medical Association. Dr. Legato was a Fellow of the American and the International Colleges of Surgeons, and had been affiliated with St. Mary's Hospital, Hoboken, and Riverside General Hospital, Secaucus. During World War II, he served with the United States Army's medical corps, commanding the medical detachment of the 44th Cavalry Reconnaissance Squadron, and was decorated for bravery. In 1981, in honor of his 50 years as a physician, Dr. Legato received MSNJ's Golden Merit Award.

Dr. John W. Marquis

At the untimely age of 59, John William Marquis, M.D., a radiologist in West Orange, died on December 24, 1985, in Overlook Hospital, Summit. Born in Texas, Dr. Marquis received his medical degree from George Washington University School of Medicine, Washington, D.C., in 1954. He was a member of our Essex County component, and of the American Medical Association. Dr. Marquis was board certified in his specialty, and a Fellow of the American College of Radiology. He had been director of radiology, a member of the board of trustees, and past president of the medical staff at East Orange General Hospital, and also had been affiliated with United Hospitals, Newark.

Dr. Richard E. Mauer

We just have learned of the death on July 2, 1985, of Richard Earl Mauer, M.D., at the age of 70. Born in Iowa, Dr. Mauer received his medical degree from the State University of Iowa's College of Medicine in 1939. A practitioner of family medicine in Rutherford for many years, Dr. Mauer retired in 1984. He had been a member of our Essex County component and of the American Medical Association, and was affiliated with St. Mary's Hospital, Passaic, and Hackensack General Hospital.

Dr. Arthur S. McCallum

A retired general practitioner and otolaryngologist, Arthur Storey McCallum, M.D., died at his home in Sanford, Florida, on November 22, 1985, at the venerable age of 87. Born in Jeffersonville, Georgia, Dr. McCallum received his medical degree from Jefferson Medical College of Philadelphia in 1922. He established his practice in Barrington where he served the surrounding communities until his retirement in 1958. Dr. McCallum was a member of our Camden County component and of the American Medical Association. He had been affiliated with St. Joseph's and University Hospitals, Philadelphia. In 1972, Dr. McCallum was a recipient of MSNJ's Golden Merit Award honoring his 50 years as a physician.

Dr. John A. McGeary

John A. McGeary, M.D., a retired specialist in obstetrics and gynecology in Essex and Union Counties, died on November 11, 1985, in Alexian Brothers Hospital, Elizabeth. Born in Jersey City in 1910, Dr. McGeary received his medical degree from Georgetown University's Medical School in 1945. He had been a member of our Union County component and of the American Medical Association. Dr. McGeary was board certified in his specialty, and his list of hospital affiliations was impressive, including Saint Barnabas Medical Center, Livingston; St. Michael's Medical Center, Newark;

and Alexian Brothers Hospital, Elizabeth General, and St. Elizabeth's Hospital, all in Elizabeth. He did medical work for Bell Laboratories in Murray Hill, and after his retirement in 1971, served as medical director of Vineland State School.

Dr. Stephen A. Mickewich

Stephen Arthur Mickewich, M.D., a Bayonne surgeon for more than 50 years, died December 17, 1985, in Bayonne Hospital. Born in 1908 in Bayonne, Dr. Mickewich received his medical degree from Loyola University Medical School in 1932. He was a member of our Hudson County component, and of the American Medical Association. Dr. Mickewich was affiliated with Bayonne Hospital, and the Medical Center and St. Francis Hospital, Jersey City. During World War II, Dr. Mickewich served as a medical officer, with the rank of major, with the United States Air Force, receiving the World War II Victory Medal and the American Theater Service Medal.

Dr. Graham C. Newbury

Graham Cyrenus Newbury, M.D., a retired surgeon, died on December 22, 1985, at Point Pleasant Hospital, at the age of 76. Born in Newark, Dr. Newbury was graduated from Yale University School of Medicine in 1935, and had practiced in the communities of Cranford, Summit, and Lavallette. He originally was a member of the our Union County component and had served as its president in 1958. Dr. Newbury later became a member of our Ocean County component, and also was a Fellow of the American College of Surgeons. He had served as chief of the surgical staff at Overlook Hospital, Summit, and later was affiliated with Point Pleasant Hospital. Dr. Newbury was civic minded: had been medical adviser for the Lavallette fire department and first aid squad; was school physician for Lavallette elementary school; and was a former member of that community's board of health. In 1985, the Medical Society of New Jersey conferred on him its Golden Merit Award in recognition of his 50 years in medicine.

Dr. Louis Pollack

Louis Pollack, M.D., an internist in Union, died on December 3, 1985, at the age of 71. A native of Brooklyn, New York, Dr. Pollack received his medical degree from the Faculty o Medicine, University of Edinburgh Scotland, in 1937. He had been member of our Union County component, and was affiliated with Alexian Brothers and Saint Elizabeth Hospitals, Elizabeth.

Dr. David L. Weiler

David Lawrence Weiler, M.D., an Oradell ophthalmologist, died on No vember 29, 1985, at the early age o 50. A native of Brooklyn, Dr. Weile was graduated from Chicago Medical School in 1960. He practiced i the states of New York and California before coming to New Jerse in 1967. He became a member of ou Bergen County component and wa board certified in his specialty. H was a Fellow of the American Colleg of Surgeons, of the America Academy of Ophthalmology an Otolaryngology, and of the New Yor Academy of Medicine. Dr. Weiler ha been affiliated with Manhattan Ey and Ear Infirmary, New York City and with Pascack Valley Hospita Westwood. He had served with th medical corps of the United State Army in Korea.

AUTHOR INFORMATION

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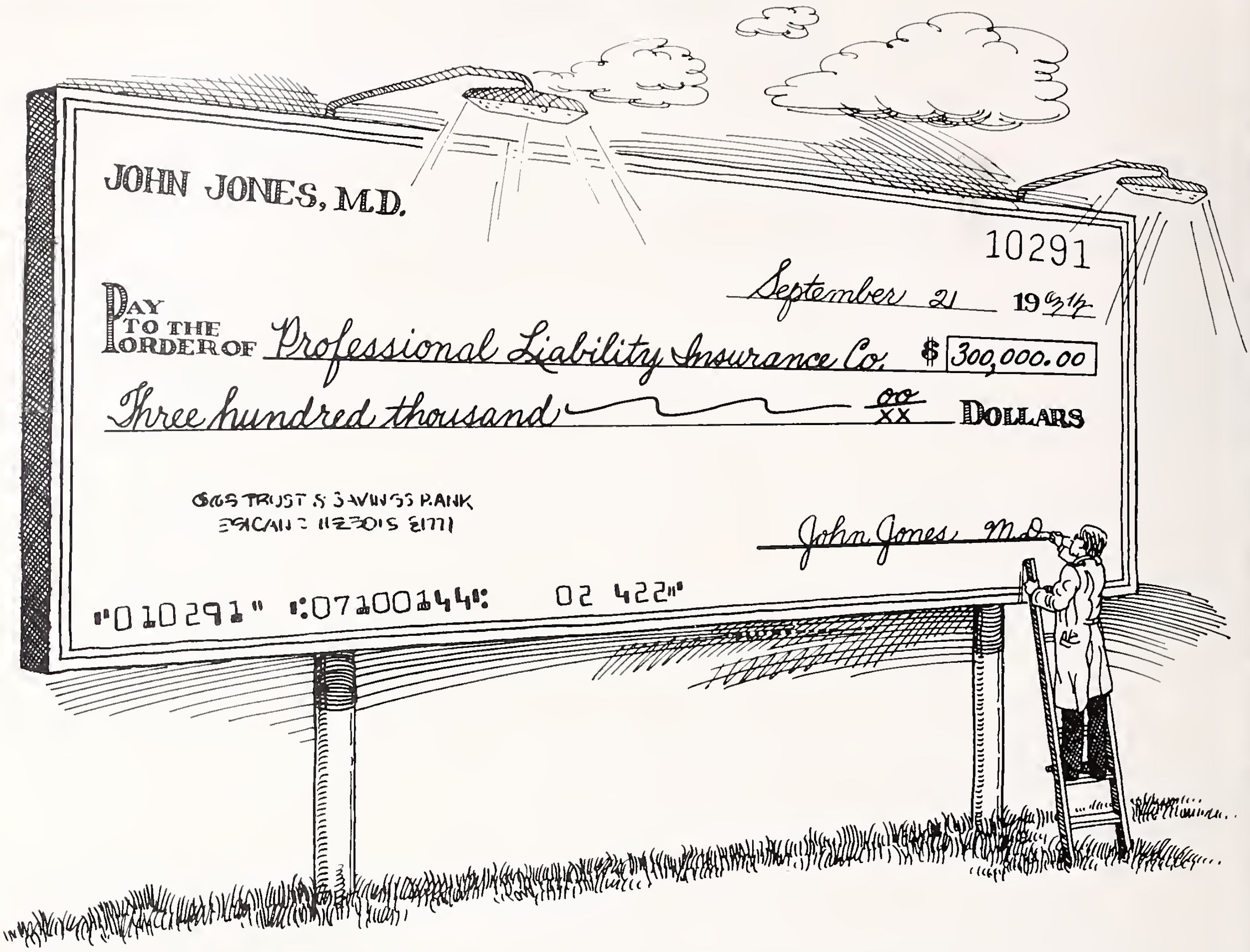
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CONTRAINDICATIONS: Hypersensitivity to trimethoprim or sulfonamides; documented megaloblastic anemia due to folate deficiency; pregnancy at term and during the nursing period; infants less than two months of age.

WARNINGS: FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS.

BACTRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION. Clinical signs, such as rash, sore throat, fever, pallor, purpura or jaundice, may be early indications of serious reactions. In rare instances a skin rash may be followed by more severe reactions, such as Stevens-Johnson syndrome, toxic epidermal necrolysis, hepatic necrosis or serious blood disorder. Perform complete blood counts frequently.

BACTRIM SHOULD NOT BE USED IN THE TREATMENT OF STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have a greater incidence of bacteriologic failure when treated with Bactrim than with penicillin.

PRECAUTIONS:

General: Give with caution to patients with impaired renal or hepatic function, possible folate deficiency and severe allergy or bronchial asthma. In glucose-6-phosphate dehydrogenase-deficient individuals, hemolysis may occur, frequently dose-related.

Information for Patients: Instruct patients to maintain adequate fluid intake to prevent crystalluria and stone formation.

Laboratory Tests: Perform complete blood counts frequently; if a significant reduction in the count of any formed blood element is noted, discontinue Bactrim. Perform urinalyses with careful microscopic examination and renal function tests during therapy, particularly for patients with impaired renal function.

Drug Interactions: In elderly patients concurrently receiving certain diuretics, primarily thiazides, an increased incidence of thrombocytopenia with purpura has been reported. Bactrim may prolong the prothrombin time in patients who are receiving the anticoagulant warfarin. Keep this in mind when Bactrim is given to patients already on anticoagulant therapy and reassess coagulation time. Bactrim may inhibit the hepatic metabolism of phenytoin. Given at a common clinical dosage, it increased the phenytoin half-life by 39% and decreased the phenytoin metabolic clearance rate by 27%. When giving these drugs concurrently, be alert for possible excessive phenytoin effect. Sulfonamides can displace methotrexate from plasma protein binding sites, thus increasing free methotrexate concentrations.

Drug/Laboratory Test Interactions: Bactrim, specifically the trimethoprim component, can interfere with a serum methotrexate assay as determined by the competitive binding protein technique (CBPA) when a bacterial dihydrofolate reductase is used as the binding protein. No interference occurs if methotrexate is measured by a radioimmunoassay (RIA). The presence of trimethoprim and sulfamethoxazole may also interfere with the Jaffé alkaline picrate reaction assay for creatinine, resulting in overestimations of about 10% in the range of normal values.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Carcinogenesis: Long-term studies in animals to evaluate carcinogenic potential not conducted with Bactrim. *Mutagenesis:* Bacterial mutagenic studies not performed with sulfamethoxazole and trimethoprim in combination. Trimethoprim demonstrated to be nonmutagenic in the Ames assay. No chromosomal damage observed in human leukocytes *in vitro* with sulfamethoxazole and trimethoprim alone or in combination; concentrations used exceeded blood levels of these compounds following therapy with Bactrim. Observations of leukocytes obtained from patients treated with Bactrim revealed no chromosomal abnormalities. *Impairment of Fertility:* No adverse effects on fertility or general reproductive performance observed in rats given oral dosages as high as 70 mg/kg/day trimethoprim plus 350 mg/kg/day sulfamethoxazole.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Trimethoprim and sulfamethoxazole may interfere with folic acid metabolism; use during pregnancy only if potential benefit justifies potential risk to fetus. Nonteratogenic Effects: See CONTRAINDICATIONS section.

Nursing Mothers: See CONTRAINDICATIONS section.

Pediatric Use: Not recommended for infants under two months (see INDICATIONS and CONTRAINDICATIONS sections).

ADVERSE REACTIONS: Most common are gastrointestinal disturbances (nausea, vomiting, anorexia) and allergic skin reactions (such as rash and urticaria). **FATALITIES ASSOCIATED WITH THE ADMINISTRATION OF SULFONAMIDES, ALTHOUGH RARE, HAVE OCCURRED DUE TO SEVERE REACTIONS, INCLUDING STEVENS-JOHNSON SYNDROME, TOXIC EPIDERMAL NECROLYSIS, FULMINANT HEPATIC NECROSIS, AGRANULOCYTOSIS, APLASTIC ANEMIA AND OTHER BLOOD DYSCRASIAS (SEE WARNINGS SECTION).** *Hematologic:* Agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, neutropenia, hemolytic anemia, megaloblastic anemia, hypoprothrombinemia, methemoglobinemia, eosinophilia. *Allergic Reactions:* Stevens-Johnson syndrome, toxic epidermal necrolysis, anaphylaxis, allergic myocarditis, erythema multiforme, exfoliative dermatitis, angioedema, drug fever, chills, Henoch-Schoenlein purpura, serum sickness-like syndrome, generalized allergic reactions, generalized skin eruptions, photosensitivity, conjunctival and scleral injection, pruritus, urticaria and rash. Periarteritis nodosa and systemic lupus erythematosus have been reported. *Gastrointestinal:* Hepatitis (including cholestatic jaundice and hepatic necrosis), elevation of serum transaminase and bilirubin, pseudomembranous enterocolitis, pancreatitis, stomatitis, glossitis, nausea, emesis, abdominal pain, diarrhea, anorexia. *Genitourinary:* Renal failure, interstitial nephritis, BUN and serum creatinine elevation, toxic nephrosis with oliguria and anuria, crystalluria. *Neurologic:* Aseptic meningitis, convulsions, peripheral neuritis, ataxia, vertigo, tinnitus, headache. *Psychiatric:* Hallucinations, depression, apathy, nervousness. *Endocrine:* Sulfonamides bear certain chemical similarities to some goitrogens, diuretics (acetazolamide and the thiazides) and oral hypoglycemic agents; cross-sensitivity may exist. Diuresis and hypoglycemia have occurred rarely in patients receiving sulfonamides. *Musculoskeletal:* Arthralgia, myalgia. *Miscellaneous:* Weakness, fatigue, insomnia.

DOSAGE AND ADMINISTRATION: Not recommended for use in infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN: *Usual adult dosage* for urinary tract infections is one DS tablet, two tablets or four teaspoonfuls (20 ml) *b.i.d.* for 10 to 14 days. Use identical daily dosage for 5 days for shigellosis. *Recommended dosage for children* with urinary tract infections or acute otitis media is 8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses every 12 hours for 10 days. Use identical daily dosage for 5 days for shigellosis. *Renal Impaired:* Creatinine clearance above 30 ml/min, give usual dosage; 15-30 ml/min, give one-half the usual regimen; below 15 ml/min, use not recommended.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS: Usual adult dosage is one DS tablet, two tablets or four teasp. (20 ml) *b.i.d.* for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS: Recommended dosage is 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

HOW SUPPLIED: *DS (double strength) Tablets* (160 mg trimethoprim and 800 mg sulfamethoxazole—bottles of 100, 250 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 20. *Tablets* (80 mg trimethoprim and 400 mg sulfamethoxazole)—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension* (40 mg trimethoprim and 200 mg sulfamethoxazole per teasp.)—bottles of 100 ml and 16 oz (1 pint). *Suspension* (40 mg trimethoprim and 200 mg sulfamethoxazole per teasp.)—bottles of 16 oz (1 pint).

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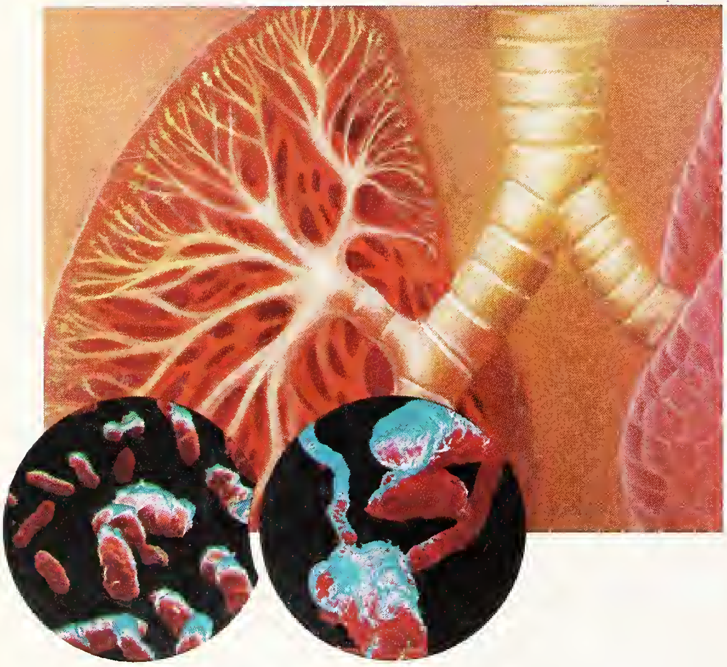

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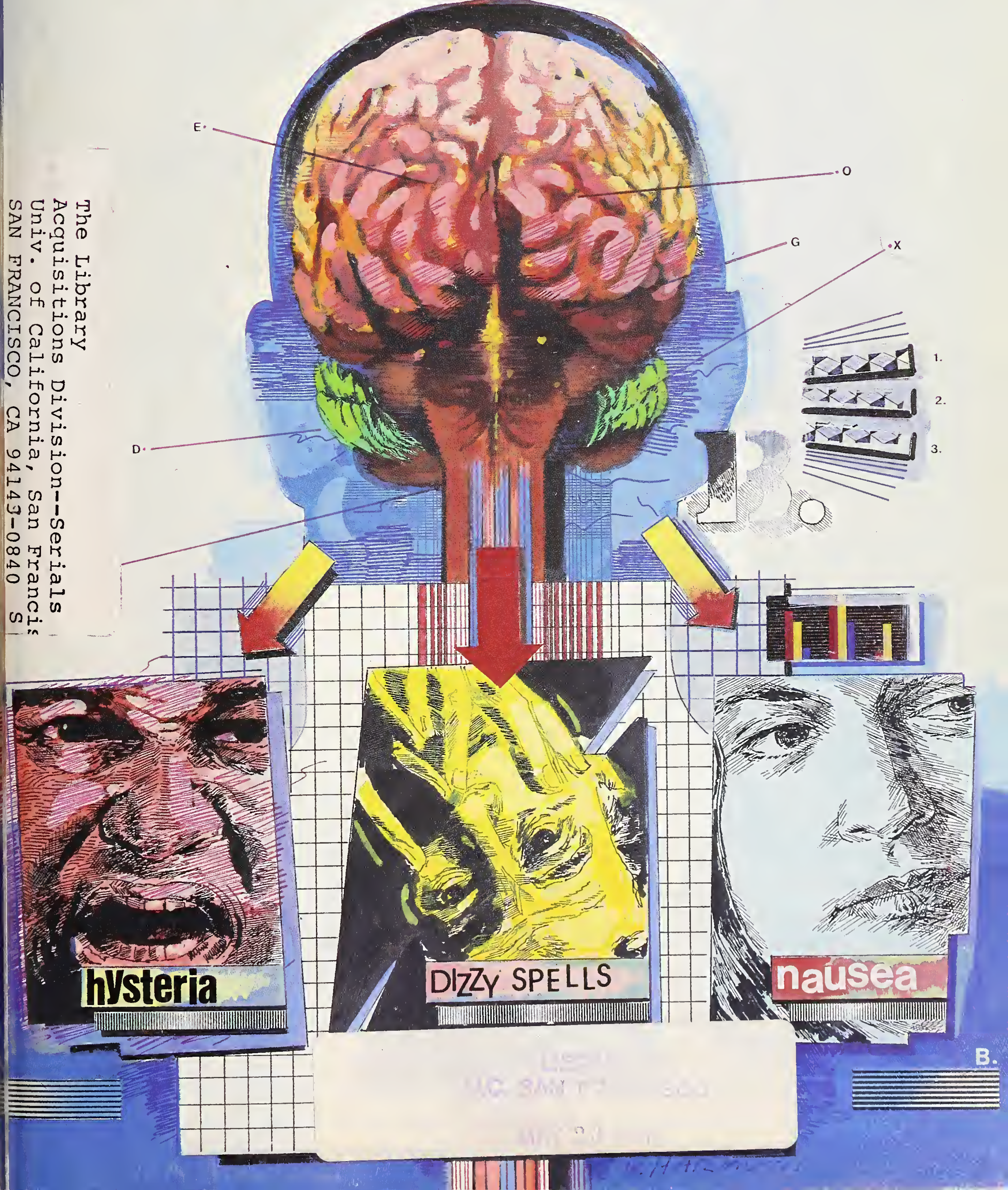

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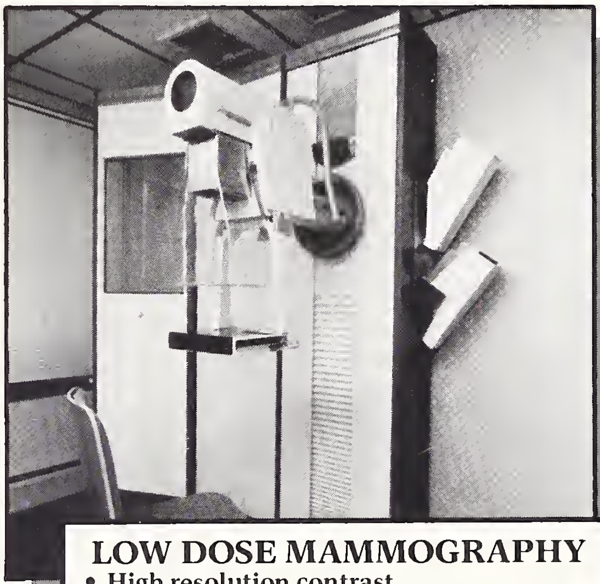

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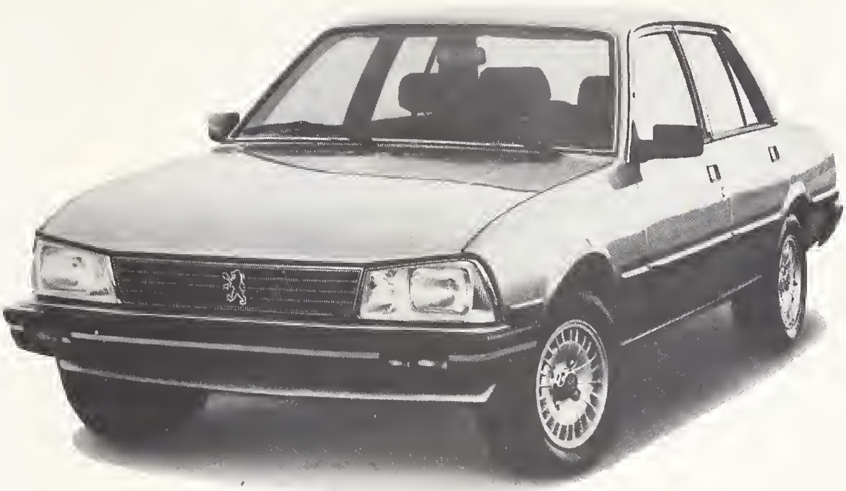

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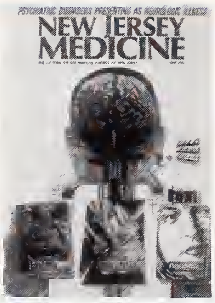

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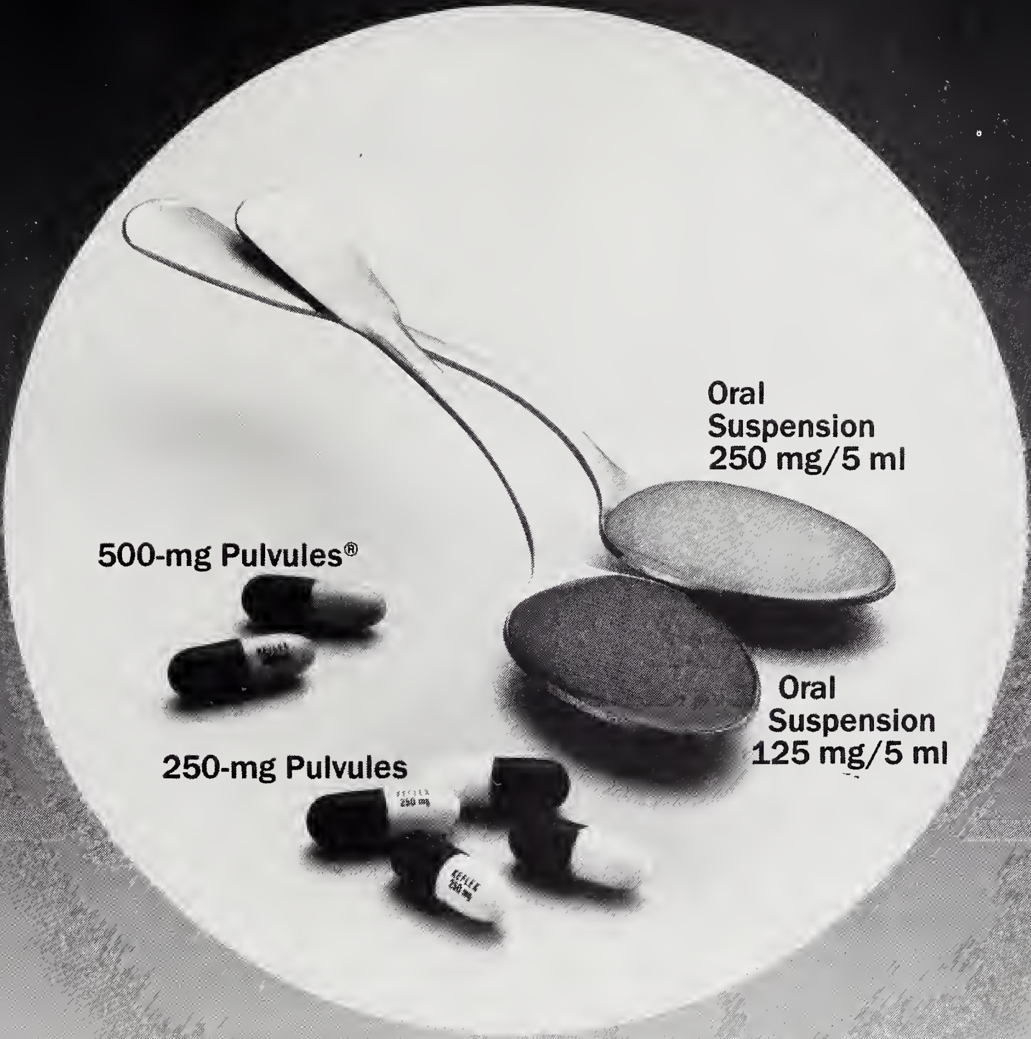

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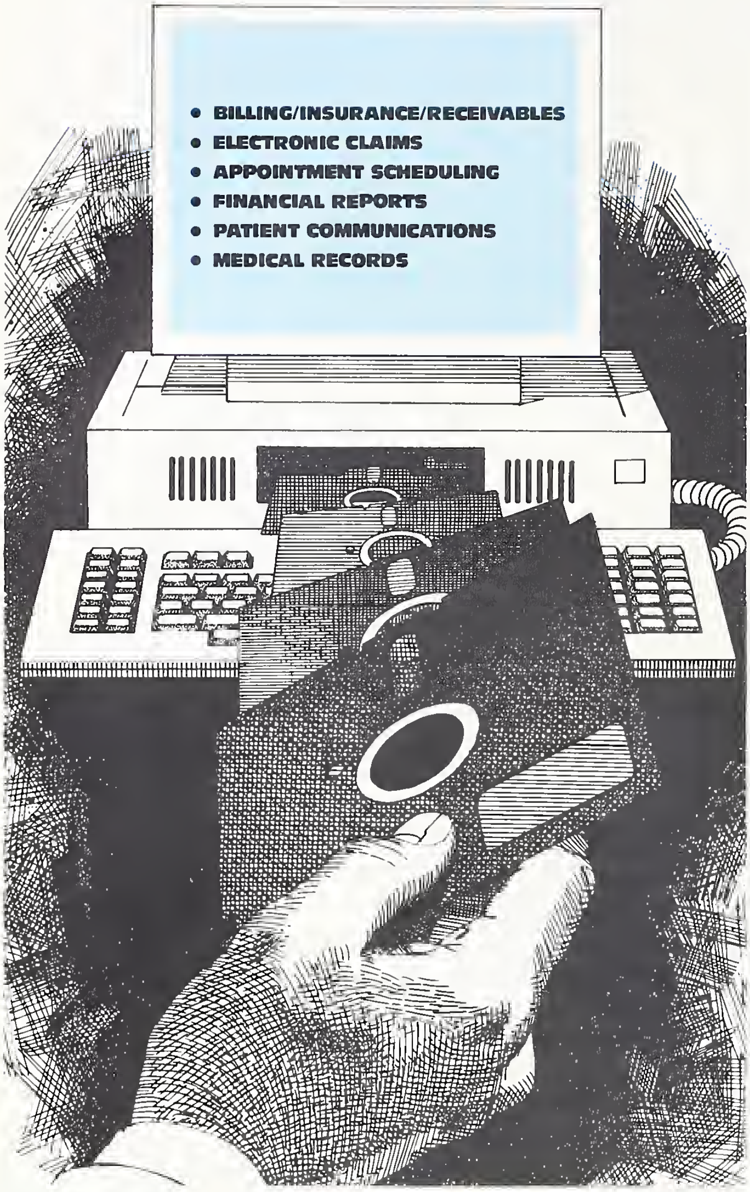

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MEMBERSHIP NEWSLETTER

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BEWARE

There has been a resurgence of a periodic effort to sell marketing services to New Jersey physicians. The promoters of these services have a variety of sales pitches to recruit physician participation, including exposure via cable TV, press releases, patient referrals via a televised telephone number, and a listing of medical who's-who via specialty.

The promoters allegedly have been indicating they have the sponsorship of the Medical Society of New Jersey. We take this opportunity to advise you this is not true.

Our sources indicate there is a charge for this promotion that varies from $500 to over $1,000, depending on the participation interval.

The Board of Medical Examiners has been advised of this promotion for its review.

DIVERS ALERT NETWORK (DAN)

The seacoast of the New Jersey shoreline affords considerable opportunity for recreational diving. Nearly 300,000 divers are certified each year. The sport has an excellent safety record but, as in all sports, there is the potential for serious injury.

The national Divers Alert Network (DAN) was established in 1980 by the Undersea Medical Society. To use the emergency Network, a diver or physician dials (919) 684-8111 and asks for DAN. The caller is connected to a diving-medicine physician at Duke University Medical Center in North Carolina available 24 hours per day. The physician may advise the caller directly or refer him to a local diving physician. If needed, the physician will work with one of the seven DAN Regional Coordinators to arrange referral and transport to an appropriate treatment facility.

All nonemergency calls should be directed to the DAN information and office center, (919) 684-2948, which is available 9 A.M. to 5 P.M., Monday through Friday.

FMG—OPINION

"We in the medical profession today face a crossroads of conscience. We have seen large numbers of new physicians enter our ranks over the past five to ten years and are now told that we have a physician surplus, a "doctor glut." Instead of accepting these new colleagues into our profession gracefully, we have put up barriers to medical staff membership, pushed for reduction in medical school class size, and now, most recently, our leaders have singled out the United States Foreign Medical Graduate (USFMG) for special attack. These, the most vulnerable of our colleagues have been targeted for extinction. Not because of any legitimate concern for quality or any genuine concern for the public welfare, but for the purpose of protecting the economic status quo of the practicing physician and the inflated budgets of our medical school bureaucracies. I raise my voice in protest. I raise my voice in anger and, indeed, in sadness at this most recent perversion of our profession produced by an alliance between our most prestigious medical institutions and a powerful group of United States senators who have been aided and abetted by elements within the present Republican administration in Washington. Some states also have joined the bandwagon to exclude United States citizens who are studying medicine abroad from returning home for graduate medical education.

"Who are the students who are forced to go elsewhere for their medical education? They, for the most part, are qualified applicants to United States schools who simply don't have room for them. Rather than take "no" for an answer, they choose to pursue their dream to become physicians.

"They study under primitive, often alien conditions, do clinical rotations in inner-city hospitals while sleeping on on-call room floors in sleeping bags because of lack of housing close to these hospitals. They represent not the worst, but the best that we can offer to the profession. They demonstrate daily by their sacrifice and their dedication to the true calling to medicine. They, the repeatedly rejected and the discarded, are still pursuing their goal, however distant, however fragile, however difficult it may be. The action by the Senate Finance Committee and the proposed action by Congressman Claude Pepper is a cynical attempt to eliminate USFMGs from graduate medical education positions and to deny them their mission. These forces are trying to save money for the budget on the backs of these idealistic and dedicated students. While political motivation may be difficult to understand, I, particularly, object when the American Medical Association, the purported representative of American medicine, endorses such a blatantly evil proposal. The AMA has been made the unwitting handmaiden of the powerful politico-economic empire of the academic medical establishment. Rather than find a way to ensure a quality medical education to all who can absorb it, they act as if their role is to protect the income of physicians from competition; rather than to display the conscience of this ancient and noble profession, they claim to represent. The inner-city hospitals that these congressional leaders wish to restrict from USFMGs serve the poorest, most vulnerable members of our society. They are the human chaff cast out to society looking for the humanity in all of us for help. The profession must return to its roots, to its original calling. We must resurrect our commitment to service, not profit, to sacrifice, not comfort, and to the conscience rather than to succumb to the short-term appeal of our politicians for economic protection at the expense of the best motivated, most dedicated of our young colleagues.

"Let us reject the easy solicitations of those who would 'protect us.' Let us once again be a part of a great and honored profession. Let us accept in our ranks, those who have been excluded too long, as long as they can pass the necessary qualifying examinations. Let us now put our money where our mouth is. Let concern for the sick, the tired, and the poor be our primary motivation. Let those forces in Washington and Chicago who would corrupt us, hear our collective refusal to be treated as another 'industry.' We are the brightest and the best. Let us return to the ancient calling of our profession. Let us be physicians, not politicians."

(signed) Fred M. Jacobs, M.D.

FINI

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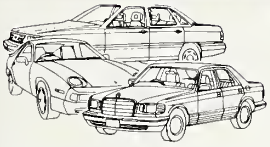

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HMO FACTS OF LIFE

By now, you've undoubtedly heard about HMOs, IPAs, et al. They're complicated, confusing and causing physicians considerable concern in terms of their present and future practices.

The following clearly defines what each is. After reviewing these various types of pre-paid health care, we believe you will have the basic information necessary to choose the best for you.

HEALTH MAINTENANCE ORGANIZATIONS (HMOs)

CENTER OR CLINIC

- Central facility or facilities
- Staff physicians
- Patient selects a primary care physician (Internist, GP, FP, and Pediatrician) from the staff
- May frequently have physician turnover resulting in short term doctor-patient relationship and/or the need to re-select primary physician

DIRECT PHYSICIAN CONTRACTING

- No central facility
- Private practicing physicians
- Patient selects a primary care physician (Internist, GP, FP, Pediatrician)
- Obstetrician-Gynecologist is not a primary, but a specialist. The patient is seen by the Obstetrician-Gynecologist only by referral from the primary care physician.
- Primary physicians are capitated. That means they are paid a fixed amount annually to treat a specific patient whether they see that patient once—or a hundred times.
- No group voice because there is no physician group
- Specialists are reimbursed a percentage of their fee
- Patients can see physicians when they want

IPA CORPORATIONS

- No central facility
- Private practicing physicians
- Patient selects 1 or 2 primary physicians: Female and male adults select an Internist, GP or FP; females additionally select an Obstetrician-Gynecologist; parents select a Pediatrician, GP or FP for their child(ren).

  Obstetrician-gynecologists are primary care physicians, and can be seen directly by the member for both routine and specialized care with no referral necessary.
- A corporate body which is organized by physicians. The IPA then contracts with an HMO to provide services to the HMO members. *The physician does not join the HMO, but simply participates.*
- The IPA is the physicians' voice to the HMO which represents the physician re: fee reimbursement, quality of care, peer review, etc.
- Traditional fee-for-service payment
- Patients can see physicians when needed. Each visit is paid for by the HMO on a fee-for-service basis

EXAMPLES

CENTER OR CLINIC	DIRECT PHYSICIAN CONTRACTING	IPA CORPORATIONS
HIP	**HMO-NJ**	**HealthWays, Inc.**
Kaiser Permanente	**HMO of Greater NY**	

If you have further questions regarding any of the above, call HealthWays' IPA Department at 1-800-624-0720 in New Jersey or 201-636-6200 out-of-state.

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PROFESSIONAL LIABILITY COMMENTARY

The Malpractice Stress Syndrome*

The impact of malpractice suits on the physician and family is called the "malpractice stress syndrome."

There has been an increasing awareness within the medical profession and among the public-at-large that malpractice litigation is part of the new American way of life. The amount of awards and the rates for coverage have been well publicized. The impact of these expenses on the cost of health care is being felt by all. The relationship of these expenses to the national economy and national debt has been part of numerous commentaries. This understanding of the malpractice issue is one centered on economics, law, and the health-care system. Attention has not been given to the issue of the person who is at the center of this complex multifaceted issue—the individual physician who is experiencing an assault on his personal and professional identity, competence, and integrity.

Doctor Sara Charles conducted two studies: physicians who were sued for malpractice in Cook County, Illinois; and sued and unsued physician-members of the Chicago Medical Society. She is coauthor, with her husband, Eugene Kennedy, of the book, *Defendant: A Psychiatrist on Trial for Medical Malpractice*, published in 1985. In Doctor Charles' studies on physicians involved in malpractice litigation, a number of significant issues have surfaced which bring to light the personal impact of malpractice stress.

Isolation. Of the physicians involved in the study, 31.4 percent felt "alone in my effort to vindicate myself"; but only 10 percent sought moral support from peers. In ways similar to how people tend to avoid persons with terminal illness, other physicians frequently avoid the physician being sued. Physicians express sorrow, sympathy, and pity for another physician being sued, but there generally is a failure to express "empathy," in part due to an aversion of facing personal vulnerability.

Negative Self-Image. Defendant physicians feel misunderstood (41.2 percent), defeated (34 percent), decreased self-confidence (31.4 percent), lower self-esteem (27.3 percent), and shame (14.4 percent).

Emotional Impact. More than 50 percent of the responding physicians in these studies experienced anger, inner tension, depressed mood, frustration, irritability, and insomnia. In one study of Cook County physicians who were sued, it was found that 39.1 percent had experienced "major depressive disorders," and 26.6 percent of the physicians had these symptoms for more than two weeks. More than 20 percent experienced pervasive anger accompanied by at least four of the following symptoms: depressed mood, inner tension, frustration, irritability, insomnia, fatigue, gastrointestinal symptoms, and headache.

Other Effects. About 36 percent of sued physicians reported difficulty in concentrating, 29 percent experienced decreased sex drive, and 27 percent noted decreased appetite. Between 5 and 8 percent developed physical illness, between 7.4 and 9 percent experienced exacerbation of previously diagnosed illness, e.g. angina, duodenal ulcer, spastic colon, and hypertension. In two studies it was found that some physicians escaped experiencing no symptoms (4.2 and 17.9 percent, respectively).

If we note all of these issues, we can see a very complex combination of physiological, emotional, and spiritual issues impacting on the physician. The ripple effect also brings a high risk to domestic stability. Additionally, the isolation experienced by the physician is experienced by the other members of the physician-family. Stress on the physician and family members is prolonged by the litigation process itself. While one study found that 50 percent of the claims filed were dropped—many remained active for 24 to 38 months.

Professional therapists have not understood fully the impact of stress caused by litigation. Further, learning how to treat the problem still is one of trial and error. For the physician who is used to treating a person with problems, it frequently is difficult for that physician to follow the adage, "Physician, heal thyself."

A new need has been recognized in the medical community. The impact of malpractice suits on the physician and the medical family can be described as "Malpractice Stress Syndrome." As has been identified with many other "stressing" diseases or situations, the Malpractice Stress Syndrome has an impact far beyond the physician. The members of the medical family most definitely are involved—and, in some cases, even more than the physician. If we take the experiences of successfully proved self-help groups, and take a look at their principles, characteristics, benefits, and practical considerations and procedures, we find guidance in establishing self-help groups for the physician and members of the medical family who are involved in "Malpractice Stress Syndrome."

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*This article was written by Rev. Edward G. Reading, Assistant Director, Impaired Physicians Program, MSNJ. This column, from the Department of Professional Liability Control, MSNJ, was prepared at the request of James E. George, M.D., J.D., and A. Ronald Rouse, who are, respectively, Director of the Department and Director of Special Projects.

In the case of malpractice stress, self-help groups help physicians realize they can live with a sense of some emotional security and stability, even when the medical practice is falling apart, and with potentially devastating press coverage. The fear associated with financial insecurity is another aspect with which a self-help group can assist. These groups provide successful "role models" within the group, making advice available from more experienced members to those of lesser experience. They provide a network of new friends and acquaintances to replace the old "non-understanding" friends and to remind the members that they are not alone. The groups provide psychological, emotional, social, and spiritual support for each other to remain healthy through an emphasis on personal satisfaction, self-esteem, productivity, and community involvement (which prevents unhealthy isolation).

The Impaired Physicians Program of the Medical Society of New Jersey works closely with a number of self-help groups, including A.A. and N.A. for alcoholics and other chemically dependent persons, Al-anon, Narc-anon, Ala-teen, and Tough Love for family members, Emotions Anonymous, Gamblers Anonymous, and suicide survivors groups. Many of our program participants are involved in I.D.A.A. (International Doctors in A.A.). Self-help groups can, and do, work. Self-help groups can be successful with helping people cope with Malpractice Stress Syndrome.

It is important to note that while the Impaired Physicians Program refers some of our physicians to these groups, we do not run them or determine their policies or procedures. Successful self-help groups need to remain independent of "outside controls."

While the Malpractice Stress Syndrome can cause physician impairment, it is prudent to try to deal with it solely through the Impaired Physicians Program at this time in history. The primary reason for this is the common misunderstanding of the concepts of "impaired physician" as alcoholism/drug abuse and addictions.

I would like to stress another primary reason to keep these "malpractice support groups" out of the domain of the Impaired Physicians Program/Committees. Because of the "still existent stigma" associated with these programs—that they are primarily for drunks and druggies—it might be used in a negative way, in the malpractice case by the attorney prosecuting the case.

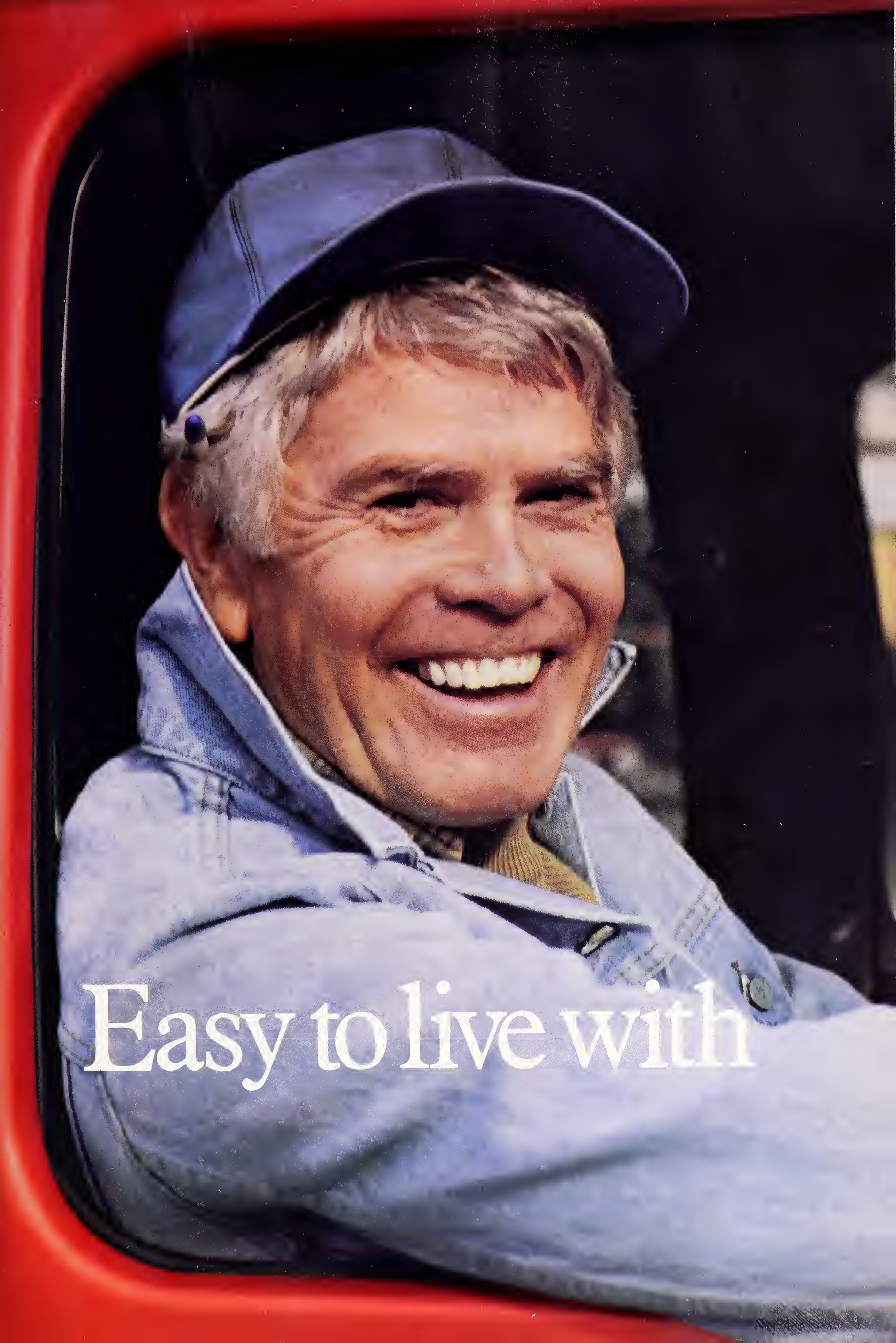
One group which is helpful in assisting medical professionals and their families in dealing with, and surviving, malpractice stress is the Litigation Stress Support Group. This group was organized in 1985 by the Auxiliary of the Medical Society of New Jersey, staff of the Medical Society of New Jersey, and the Medical Inter-Insurance Exchange of New Jersey. The Impaired Physicians Program provides assistance in the beginning stages of group development as a "prevention effort" for dealing with physician impairment. The group is based on self-help, or mutual-help principles which provide peer-group support. The group helps the participants deal with the personal, family, and professional stresses which surface when malpractice litigation becomes an "active participant" in the life of the medical family.

The group is not a lobby group, it is not a peer group for individual cases, and it is not a professional therapy group (although it can be therapeutic). The group is one which says that "You are not alone"; "Others have gone through the same thing you're going through, and they've survived"; and "So can you!"

Each meeting is divided into two parts. The first is educational in nature about some aspects of litigation, family living, and dealing with stress. The second part is a group discussion based on the principles of mutual self-help. Each meeting lasts approximately 90 minutes.

Participation in the group is open to medical professionals who are in the process of malpractice litigation, spouses/family members/significant others of medical professionals who are involved in litigation; and medical professionals/family members who presently are not involved in any litigation but have been through it and survived, and who are willing to share their experiences and offer strength and hope to those presently involved.

Participation in the group is strictly confidential; there is no official membership list. Many members are available through a telephone network to "talk" one-to-one, till the next group meeting. The group meets once a month. To find out the time and place of the next meeting, call the Medical Society of New Jersey Auxiliary at 609-896-1766.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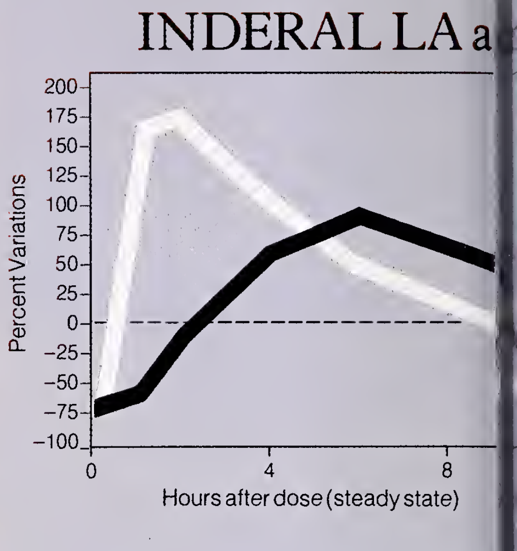

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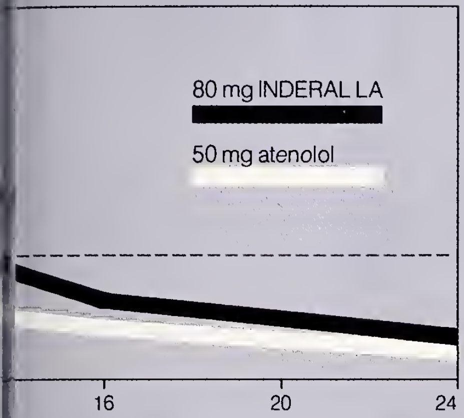

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CONTRAINDICATIONS

Propranolol hydrochloride (INDERAL® LA): Propranolol is contraindicated in: 1) cardiogenic shock; 2) sinus bradycardia and greater than first degree block; 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with propranolol.

Hydrochlorothiazide: Hydrochlorothiazide is contraindicated in patients with anuria or hypersensitivity to this or other sulfonamide-derived drugs.

WARNINGS

Propranolol hydrochloride (INDERAL® LA): CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated, and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or propranolol should be discontinued (gradually, if possible).

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction following *abrupt* discontinuance of propranolol therapy. Therefore, when discontinuance of propranolol is planned the dosage should be gradually reduced and the patient carefully monitored. In addition, when propranolol is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If propranolol therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute propranolol therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD, IN GENERAL, NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution, since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

DIABETES AND HYPOGLYCEMIA: Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin. Hypoglycemic attacks may be accompanied by a precipitous elevation of blood pressure.

Hydrochlorothiazide: Thiazides should be used with caution in severe renal disease. In patients with renal disease, thiazides may precipitate azotemia. In patients with impaired renal function, cumulative effects of the drug may develop.

Thiazides should also be used with caution in patients with impaired hepatic function or progressive liver disease, since minor alterations of fluid and electrolyte balance may precipitate hepatic coma.

Thiazides may add to or potentiate the action of other antihypertensive drugs. Potentiation occurs with ganglionic or peripheral adrenergic-blocking drugs.

Sensitivity reactions may occur in patients with a history of allergy or bronchial asthma.

The possibility of exacerbation or activation of systemic lupus erythematosus has been reported.

PRECAUTIONS

Propranolol hydrochloride (INDERAL® LA): GENERAL: Propranolol should be used with caution in patients with impaired hepatic or renal function. Propranolol is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that propranolol may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs, such as reserpine should be closely observed if propranolol is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity, which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies, in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. Propranolol has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximal recommended human dose. There are no adequate and well-controlled studies in pregnant women. Propranolol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

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NURSING MOTHERS: Propranolol is excreted in human milk. Caution should be exercised propranolol is administered to a nursing mother.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

Hydrochlorothiazide: GENERAL: Periodic determination of serum electrolytes to c possible electrolyte imbalance should be performed at appropriate intervals.

All patients receiving thiazide therapy should be observed for clinical signs of fluid or elect imbalance, namely: Hyponatremia, hypochloremic alkalosis, and hypokalemia. Serum and electrolyte determinations are particularly important when the patient is vomiting excessiv receiving parenteral fluids. Medication such as digitalis may also influence serum electro Warning signs irrespective of cause are: Dryness of mouth, thirst, weakness, lethargy, drowsi restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardi gastrointestinal disturbances such as nausea and vomiting.

Hypokalemia may develop, especially with brisk diuresis, when severe cirrhosis is prese during concomitant use of corticosteroids or ACTH.

Interference with adequate oral electrolyte intake will also contribute to hypokalemia. H kalemia can sensitize or exaggerate the response of the heart to the toxic effect of di (eg, increased ventricular irritability). Hypokalemia may be avoided or treated by use of potas supplements, such as foods with a high potassium content.

Any chloride deficit is generally mild and usually does not require specific treatment, e under extraordinary circumstances (as in liver or renal disease). Dilutional hyponatremia may in edematous patients in hot weather; appropriate therapy is water restriction, rather than adr tration of salt, except in rare instances when the hyponatremia is life-threatening. In actua depletion, appropriate replacement is the therapy of choice.

Hyperuricemia may occur or frank gout may be precipitated in certain patients receiving thi therapy.

Insulin requirements in diabetic patients may be increased, decreased, or unchanged. Dia mellitus which has been latent may become manifest during thiazide administration.

If progressive renal impairment becomes evident, consider withholding or discontinuing di therapy.

Thiazides may decrease serum PBI levels without signs of thyroid disturbance.

Calcium excretion is decreased by thiazides. Pathologic changes in the parathyroid glan hypercalcemia and hypophosphatemia have been observed in a few patients on prolo thiazide therapy. The common complications of hyperparathyroidism, such as renal lithiasis, resorption, and peptic ulceration, have not been seen. Thiazides should be discontinued b carrying out tests for parathyroid function.

DRUG INTERACTIONS: Thiazide drugs may increase the responsiveness to tubocurarine.

The antihypertensive effects of thiazides may be enhanced in the postsympathectomy pa Thiazides may decrease arterial responsiveness to norepinephrine. This diminution is not suff to preclude effectiveness of the pressor agent for therapeutic use.

PREGNANCY: Pregnancy Category C. Thiazides cross the placental barrier and appear i blood. The use of thiazides in pregnancy requires that the anticipated benefit be weighed ag possible hazards to the fetus. These hazards include fetal or neonatal jaundice, thrombocytop and possibly other adverse reactions which have occurred in the adult.

NURSING MOTHERS: Thiazides appear in human milk. If use of the drug is deemed ess the patient should stop nursing.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Propranolol hydrochloride (INDERAL® LA): Most adverse effects have been mil transient and have rarely required the withdrawal of therapy.

Cardiovascular: Bradycardia; congestive heart failure; intensification of AV block; hypoter paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Ray type.

Central Nervous System: Lightheadedness; mental depression manifested by inso lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; disturbances; hallucinations; an acute reversible syndrome characterized by disorientati time and place; short-term memory loss; emotional lability; slightly clouded sensorium decreased performance on neuropsychometrics.

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, c pation; mesenteric arterial thrombosis; ischemic colitis.

Allergic: Pharyngitis and agranulocytosis; erythematous rash; fever combined with achin sore throat; laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis; nonthrombocytopenic purpura, thrombocytopenic purpu

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been repor

Miscellaneous: Alopecia; LE-like reactions; psoriasiform rashes; dry eyes; male impotence Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the serous membranes, and conjunctivae reported for a beta blocker (practolol) have not associated with propranolol.

Hydrochlorothiazide:

Gastrointestinal: Anorexia; gastric irritation, nausea, vomiting, cramping, diarrhea, constip jaundice (intrahepatic cholestatic jaundice); pancreatitis; sialadenitis.

Central Nervous System: Dizziness, vertigo; paresthesias; headache; xanthopsia.

Hematologic: Leukopenia; agranulocytosis; thrombocytopenia; aplastic anemia.

Cardiovascular: Orthostatic hypotension (may be aggravated by alcohol, barbiturate narcotics).

Hypersensitivity: Purpura; photosensitivity; rash; urticaria; necrotizing angiitis (vasc cutaneous vasculitis); fever; respiratory distress, including pneumonitis; anaphylactic reacti

Other: Hyperglycemia; glycosuria; hyperuricemia; muscle spasm; weakness; restless transient blurred vision.

Whenever adverse reactions are moderate or severe, thiazide dosage should be reduc therapy withdrawn.

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REFERENCES

1. Data on file, Ayerst Laboratories. **2.** Ravid M, Lang R, Jutrin I: The relative antihyperte potency of propranolol, oxprenolol, atenolol, and metoprolol given once daily. *Arch Intern* 1985;145:1321-1323. **3.** Sumiye L, Vivian AS, Frisof KB, et al: Potassium loss associate hydrochlorothiazide versus chlorthalidone. *Clin Ther* 1981;4:308-320. **4.** Ram CVS, Garret Kaplan NM: Moderate sodium restriction and various diuretics in the treatment of hyperte *Arch Intern Med* 1981;141:1015-1019.

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A Tribute and a Comment

Armando F. Goracci, M.D., the 189th President of the Medical Society of New Jersey, died on February 7, 1986.

The Medicare assignment program seems to be shortchanging participating physicians.

Armando F. Goracci has died and left a large vacuum on this earth and in the hearts of his family and friends. Those of us who knew him and worked for and with him had a quiet admiration for this gentle giant of a man. Kindness, consideration, and gratitude exuded from him every time I was in his presence.

He was the 189th President of the Medical Society and much more.

In his Farewell Address, "The Year of Challenges," Dr. Goracci spoke about DRGs, FMGs, fee schedules, schedule II drugs, and prescribing procedures. He worried about impaired physicians, cutbacks in federal funding, and persons "who may not be able to procure proper medical care by reason of inadequate health insurance benefits." He was alarmed at the rising costs of health care and said: "Every effort must be expended to hold the cost down without sacrificing quality and availability of care for all patients."

His grateful nature was clearly evident in his expression of thanks to Vincent Maressa and the MSNJ staff. He offered "very special thanks to my wife, Connie, and my two partners—Thomas Mervine and Steven Nicoll." He ended the piece with the finale: "I want to thank the House of Delegates for allowing me to be President of the Society."

Armando's surgical skills, high professional ideals, and leadership qualities are how we are likely to remember him, but if that is all, we have missed his humanity.

Armando, on behalf of the House of Delegates, the Board of Trustees, the officers, and staff, I want to thank you for allowing us to have had you as President of the Society. We pray that you have peace in your eternal sleep.— A.K.

MEDICARE ASSIGNMENT

Robert A. Goldstone, M.D.*

Not too long ago, the government entered into a solemn contract with physicians who agreed to accept Medicare assignment, and become "participating physicians." For their cooperation, they were promised preferential treatment in the fall of 1985 when the prevailing fee schedule was to be permitted to rise.

This promise was betrayed, and fees were frozen.

Now, under the mandate of the Gramm-Rudman-Hollings bill, participating physicians have received "preferential" penalties. While participating physicians may not pass the 1 percent reduction on to their patients, nonparticipating physicians may. Certainly, this is a betrayal of the promise that the government would treat participating physicians favorably.

While it is futile to argue with the provisions of the deficit control act, it is entirely reasonable to demand that participating physicians be permitted to pass this additional expense on to the patient.

Steadily increasing rents, salaries, and sky-rocketing malpractice premiums, do not permit the already short-changed participating physician to pay a further penalty. We have been rewarded for placing our trust in our government's word by a betrayal which has verified that we participating physicians are the fools that our nonparticipating colleagues perceived us to be from the beginning.

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TRAVEL BY AIRPLANE DURING PREGNANCY

JAMES L. BREEN, M.D., CATERINA A. GREGORI, M.D., ROBERT N. NEILSON, M.D., LIVINGSTON*

Travel by airplane during pregnancy is not uncommon. This article reviews the subject and points out some inherent hazards that are present for the pregnant female.

Travel during pregnancy has long been a concern to the obstetrician. In the days of old, travel by horse and buggy was not recommended for pregnant women, but that method has given way to trains, automobiles, buses, and airplanes. By the year 2000, travel via space shuttle may concern the practicing obstetrician.

In the 1980s, outside of automobile travel of short and medium distances, airplane travel is the most common form of transportation in the United States for longer distances. Pregnant patients often travel by air for business, vacations, or visits with relatives. Commercial airplane travel has become so easy that it generally is accepted as safe and the mode of choice for long-distance travel for the pregnant female.

The obstetrician, however, should be aware of the physiologic changes and direct effects that occur during flight so he may counsel patients appropriately.

HISTORY OF TRAVEL AND PREGNANCY

Travel, of any type, during pregnancy was a major concern due to the possibility of spontaneous abortion until the end of World War II, when it became necessary to transport large numbers of people. The United States Armed Services kept track of pregnant women being transported by various means, over various distances, and during different stages of pregnancy. There was general agreement that travel, regardless of mode, time of pregnancy, or distance, had no adverse effects on pregnancy.[1-4] In fact, the rate of spontaneous abortion was less in the groups that traveled than those that did not travel.[1-4] The group included a subset who traveled by air, averaging 10 to 15 percent of the individuals studied. Again, no ill effects were noted and there was no increased incidence of spontaneous abortion.[1-4] These studies provided a basis for obstetricians to approve travel during pregnancy, including travel by commercial airlines.

PHYSIOLOGIC EFFECTS OF FLYING

Motion Sickness: This problem is common to many persons on all forms of transportation, but appears to be frequent during flight. Women in the early stages of pregnancy experiencing "morning sickness" often find flying aggravates this condition. If air turbulence is encountered, the frequency of air sickness is increased. To minimize this problem, patients should be seated midplane, near the window, partially reclining and flying at night when possible.[6] Anti-nauseant drugs may be used in severe cases. Good choices would include compazine, trimethobenzamide HCl, and scopolamine patches, since these medicines have been used extensively in pregnancy.[5]

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Acceleration, Deceleration, Turbulence, and Trauma: The major risk of these problems relates to trauma.[6] As pregnancy advances, the enlarging abdomen becomes cumbersome, balance becomes poor, and movements somewhat awkward.[7] Pregnant women moving in the aisle pose a danger to themselves and other passengers. In general, a seat near a bulkhead should be booked since there usually is an open space which provides more leg room. Pregnant women should wear their seat belts. Patients less than 12 weeks pregnant should wear the seat belt as in the nonpregnant state since the uterus is within the bony pelvis. After this time, the belt should not be worn across the abdomen due to the possibility of inflicting trauma to the uterus. It should be placed low, either at the groin level, or across the upper thighs.[7,8] Pregnant patients also should be cautioned about prolonged immobilization during a long flight. Venous pooling in the lower extremities is exaggerated in pregnancy due to uterine compression of the vena cava, and may lead to peripheral edema. The economy class seating in most airlines aggravates this problem,[8] which may be made worse by the decreased atmospheric pressure that exists in commercial airplanes. Venous stasis increases the risk of embolic disease.[9] On long flights, pregnant women should walk about every one to two hours to minimize this problem.[6,8] When edema does occur, it will require several days to resolve.

Patients with a complicated pregnancy, multiple gestation, or those late in pregnancy should not be permitted to fly except in unusual circumstances.

Decreased Humidity: The cabin humidity of most commercial airplanes is maintained at 8 percent[7,10] which is significantly less than at sea level and causes drying of mucus membranes and increased water loss. During short flights, drinking 250 ml of liquid other than coffee, tea, or alcohol[6] will compensate for the water loss.[9] Low humidity and decreased cabin pressure[6] also may cause "red eye" to which pregnant women appear to be sensitive.[8] Artificial tears can be very helpful in alleviating eye irritation during flight.[6] Pregnant women should be advised to maintain fluid intake on long flights and be cautioned against the intake of alcohol whose effects are doubled due to the physiologic changes of flight.[8]

Circadian Dysrhythmia: Transmeridianal dysrhythmia or jet lag occurs when times zones are crossed rapidly in an east-west or west-east direction. Research by the National Aeronautics and Space Administration has shown this to be related to desynchronization of the endogenous biological clock of the human being.[8] This occurs with movement through at least three time zones. Jet lag is marked by extreme fatigue, impairment of manual dexterity, and poor mental functioning.[5,6,8] Sleep, appetite, and body temperature cycles are disturbed.[8] As the pregnancy progresses, fatigue and jet lag increase. Major decision making and much activity should be postponed for at least 48 hours after arrival.[5,8] As eating and sleep cycles adjust to the day-night schedule, fatigue will lessen and mental awareness will sharpen. The effect of jet lag can be reduced by slowly adjusting eating and sleeping habits to the destination point several days before leaving.[5,8]

Hypoxia: Commercial jetliners are only partially pressurized at a constant of 8.6 pounds per square inch to the outside pressure.[9] Most commercial flights are made at an altitude between 30,000 to 45,000 feet. The cabin pressure is equivalent to 5,500 to 9,000 feet sea level with the average being 7,500 feet. The average individual can tolerate 10,000 feet sea level without showing significant signs of air hunger.[10]

But what about the pregnant female and her fetus? Luft and Finkelstein studied the "oxygen cascade" from the inspired air to the tissues at sea level, 5,300, and 8,000 feet.[11] Their results showed a mean capillary partial pressure of oxygen of 60 mm Hg at sea level and 46 mm Hg at 8,000 feet. In 1962, Wuft studied the effect of this reduced available oxygen on the fetus.[12] He permitted pregnant women to breath air with 21 percent and 15 percent oxygen and measured the fetal umbilical vein oxygen level. Air at 15 percent oxygen is equivalent to just under 8,000 feet sea level. The umbilical P02 at 8,000 feet was found to be 25.6 mm Hg. This is only 5.6 mm Hg difference from the umbilical P02 of a pregnant woman at sea level. This narrowing of the difference between the partial pressure of oxygen at sea level and 8,000 feet from the inspired air (40 mm Hg) to umbilical (5.6 mm Hg) is explained by shifts in the oxy-hemoglobin.[19] These compensatory mechanisms minimize the change in umbilical vein oxygen levels to the extent that there is no adverse effect on the fetus.[10] It should be noted that hypoxia is not a concern when flying in the supersonic transport, which flies at 60,000 feet and is fully pressurized to sea level.[8]

EMERGENCY AIR AMBULANCE TRANSPORT

The small planes used for emergency air transport rarely fly higher than 15,000 feet and are pressurized to an altitude of 5,000 to 8,000 feet. Here, mild hypoxia becomes a significant problem.[13] Complicated obstetrical patients may require air transportation to a tertiary care center. In cases of pre-eclampsia, abruptio placenta, concealed abruption, chronic hypertension, and intrauterine growth retardation, placental function already is compromised significantly.[13] Oxygen transfer is borderline at best, so the added hypoxia of 8,000 feet would create a hypoxic state for an already jeopardized fetus. Also, patients with premature labor on Ritodrine® tocolytic therapy would be at risk for two reasons: the stress of labor and the fact that Ritodrine® has the potential to interfere with the compensatory mechanisms that maintain uterine blood flow in the face of mild hypoxia.[13] Also, obstetrical patients with anemia in the range of 7 to 8 gm of Hb/dl would be affected significantly by the mild hypoxia of air transport.[6] Patients requiring air transport under these conditions should have oxygen by face mask to bring the inspired partial pressure of oxygen to 200 mm Hg.[13]

INFLIGHT EMERGENCIES OF PREGNANT WOMEN

The official statement by the International Air Transport Association (IATA), adopted by most airlines, is that on domestic flights, pregnant women can travel freely up to the 36th week. On overseas flights, the cutoff is 35 weeks. If a patient must fly beyond this point, her doctor's permission is required. The attending physician must write a letter explaining the patient's condition and complete a medical form for the airline.

Airlines are classified as "common carriers" in the United States,[9] and are not required to attend to the medical problems of passengers. The first aid kits are very basic, including only bandages. The only medications are aspirin, acetaminophen, and antiemetics. The only instrument available is a flashlight. Oxygen is available through the aircraft's built-in system and is supplied at a rate of 4 liters/minute. There is no requirement for medically trained personnel. Flight attendants are usually trained in basic first aid (4 to 12 hours) and report medical emergencies to the captain.[9] The captain can establish ground communication with an identified medical resource or the company's flight surgeon. In severe cases, flights can be diverted to the nearest airport for emergency landing. The nearest terminal usually is within 30 to 40 minutes on domestic flights. All of the above should be kept in mind when permitting a pregnant patient to fly beyond 35 to 36 weeks' gestation.

SUMMARY

Commercial air travel is quick, relatively comfortable, and generally safe for pregnant patients. In spite of this, physicians should not take such travel lightly, since there are risks for the pregnant patient. Patients with a complicated pregnancy, multiple gestation, or those late in pregnancy should not be permitted to fly except in extremely unusual circumstances.

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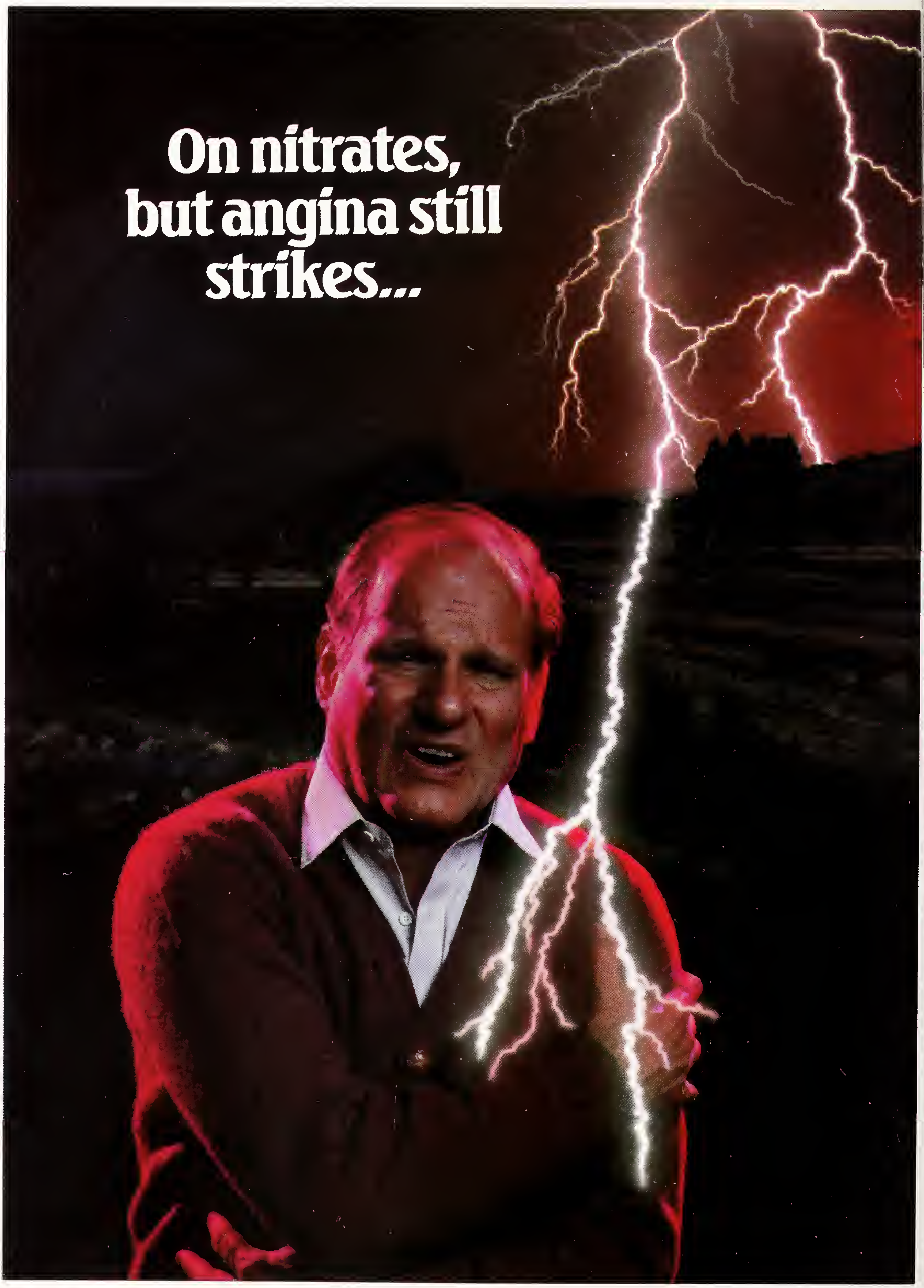
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Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Such elevations may disappear even with continued treatment; however, four cases of hepatocellular injury by verapamil have been proven by rechallenge. Periodic monitoring of liver function is prudent during verapamil therapy. Patients with atrial flutter or fibrillation and an accessory AV pathway (e.g. W-P-W or L-G-L syndromes) may develop increased antegrade conduction across the aberrant pathway bypassing the AV node, producing a very rapid ventricular response after receiving ISOPTIN (or digitalis). Treatment is usually D.C.-cardioversion, which has been used safely and effectively after ISOPTIN. Because of verapamil's effect on AV conduction and the SA node, 1° AV block and transient bradycardia may occur. High grade block, however, has been infrequently observed. Marked 1° or progressive 2° or 3° AV block requires a dosage reduction or, rarely, discontinuation and institution of appropriate therapy depending upon the clinical situation. Patients with hypertrophic cardiomyopathy (IHSS) received verapamil in doses up to 720 mg/day. It must be appreciated that this group of patients had a serious disease with a high mortality rate and that most were refractory or intolerant to propranolol. A variety of serious adverse effects were seen in this group of patients including sinus bradycardia, 2° AV block, sinus arrest, pulmonary edema and/or severe hypotension. Most adverse effects responded well to dose reduction and only rarely was verapamil discontinued. **Precautions:** ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacologic effects. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, close surveillance of vital signs and clinical status should be carried out. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patients should be carefully monitored to avoid over- or under-digitalization. ISOPTIN may have an additive effect on lowering blood pressure in patients receiving oral antihypertensive agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Clinical experience with the concomitant use of ISOPTIN and short- and long-acting nitrates suggest beneficial interaction without undesirable drug interactions. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. *Pregnancy Category C*: There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use. **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia: HR < 50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%), elevations of liver enzymes have been reported. (See *Warnings.*) The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: ecchymosis, bruising, gynecomastia, psychotic symptoms, confusion, paresthesia, insomnia, somnolence, equilibrium disorder, blurred vision, syncope, muscle cramp, shakiness, claudication, hair loss, macules, spotty menstruation. **How Supplied:** ISOPTIN (verapamil HCl) is supplied in round, scored, film-coated tablets containing either 80 mg or 120 mg of verapamil hydrochloride and embossed with "ISOPTIN 80" or "ISOPTIN 120" on one side and with "KNOLL" on the reverse side. Revised August, 1984. 2385

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DIAGNOSIS AND TREATMENT OF POPLITEAL ARTERY ANEURYSMS

WILLIAM L. DIEHL, M.D., AND GHASSEM NIKNEJAD, M.D., MORRISTOWN*

We found 22 popliteal artery aneurysms in 14 male patients over a six-year period. Most were asymptomatic and 12 patients had aneurysms elsewhere. Hypertension, coronary artery disease, and diabetes were found. Sixteen aneurysms were bypassed, 1 needed amputation, and 5 were observed.

Popliteal artery aneurysms are the most common peripheral aneurysm and are prone to a high rate of complications which endanger the viability of the involved extremity. We have sound surgical methods to correct this condition, but the condition goes undetected until complications occur. Even then, the origin of these complications often is not recognized and inappropriate therapy may be initiated.

MATERIALS AND METHODS

A retrospective chart review of popliteal aneurysms treated at Morristown Memorial Hospital from June 1977 to December 1982 was conducted. We considered age, sex, clinical presentation, associated aneurysms, concomitant disease, and type of therapy.

RESULTS

Fourteen patients with 22 documented popliteal aneurysms were treated. All patients were male between 53 and 98 years of age (average age, 70). Ten patients were older than 65 years old and 4 were younger. Eight patients (57 percent) had bilateral popliteal aneurysms. The right and left sides were affected equally (11 for each side). Twelve patients (86 percent) had additional extra-popliteal aneurysms at different levels: 8 aorto-iliac aneurysms (86 percent), 3 femoral aneurysms, (25 percent), and 1 iliac aneurysm (8 percent).

The size of these aneurysms was determined from the arteriograms or the physician's clinical estimate. On that basis, 11 aneurysms were categorized as "small" (3 cm or less in diameter) and 7 were considered to be "large" (greater than 3 cm). The size of 4 aneurysms could not be determined. Fourteen aneurysms (64 percent) were patent and 8 (36 percent) were thrombosed at the time of detection; 3 were small, 2 were large, and 3 were of undetermined size.

Seventeen aneurysms (77 percent) were symptomatic and 5 (23 percent) were without symptoms, being detected upon examination of the contralateral popliteal space. Symptoms varied from acute limb ischemia in five patients (29 percent), distal embolization with digital ischemia in four (24 percent), intermittent claudication in four (24 percent), presence of a popliteal mass in two (12 percent), pain in the popliteal space in one (6 percent), and loss of balance in the remaining patient (6 percent).

Associated conditions were seen in order of frequency as follows: hypertension in five patients (36 percent), coronary artery disease in four patients (28 percent); three patients (21 percent) had diabetes mellitus; and one patient had cirrhosis with portal hypertension and variceal bleeding.

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TABLE 1

Clinical Features

Author	Year	No. of Patients	Males	No. of Cases	Bilateral	Asymptom.	Thrombosed
Whitehouse[26]	1983	61	59	88	27 (44%)	40 (45%)	21 (24%)
Reilly[13]	1983	159	149	244	85 (53%)	112 (45%)	89 (36%)
Vermilion[12]	1981	87	84	147	60 (68%)	49 (33%)	66 (45%)
Szilagyi[2]	1981	62	62	87	28 (45%)	37 (42%)	?
Guvendik[1]	1980	21	21	28	7 (33%)	7 (33%)	9 (33%)
Inahara[3]	1978	30	28	44	14 (47%)	17 (43%)	17 (43%)
Chitwood[18]	1978	26	25	35	9 (33%)	9 (25%)	?
Tompkins[16]	1977	18	17	26	8 (45%)	7 (28%)	16 (61%)
Alpert[27]	1977	43	41	64	21 (49%)	4 (9%)	?
Towne[17]	1976	80	75	119	39 (48%)	17 (21%)	?
Hardy[28]	1975	23	21	31	8 (35%)	3 (13%)	11 (35%)
Evans[29]	1971	36	36	56	20 (55%)	17 (30%)	20 (35%)
Wychulis[14]	1970	152	148	233	89 (59%)	54 (23%)	65 (28%)
Baird[6]	1966	36	33	51	15 (42%)	12 (24%)	22 (44%)
Edmunds[30]	1965	82	81	98	26 (31%)	23 (23%)	23 (23%)
Present Study	1983	14	14	22	8 (57%)	23 (5%)	8 (36%)
Total		930	894	1373	464 (50%)	431 (31%)	367 (34%)

TABLE 2

Associated Aneurysms

Author	No. of Patients	Patients with Other Aneurysm	Aorta	Iliac	Femoral
Whitehouse[26]	61	41 (67%)	38 (62%)	22 (36%)	23 (38%)
Reilly[13]	159	70 (44%)	34 (49%)	13 (19%)	23 (32%)
Vermilion[12]	87	44 (55%)	35 (72%)	22 (47%)	30 (62%)
Szilagyi[2]	62	40 (64%)	25 (62%)	— —	27 (68%)
Guvendik[1]	21	6 (29%)	6 (100%)	— —	— —
Inahara[3]	30	19 (63%)	11 (58%)	3 (16%)	5 (26%)
Chitwood[18]	26	11 (42%)	8 (73%)	1 (18%)	2 (9%)
Tompkins[16]	18	6 (33%)	5 (83%)	3 (50%)	3 (50%)
Alpert[27]	43	11 (26%)	4 (36%)	5 (72%)	8 (45%)
Towne[17]	80	30 (31%)	15 (50%)	15 (50%)	14 (25%)
Hardy[28]	23	10 (43%)	?	?	?
Evans[29]	36	13 (36%)	13 (100%)	5 (38%)	5 (38%)
Wychulis[14]	152	69 (45%)	53 (78%)	30 (44%)	44 (65%)
Baird[6]	36	8 (22%)	6 (75%)	7 (87%)	4 (50%)
Edmunds[30]	82	12 (15%)	?	?	?
Present Study	14	12 (86%)	8 (66%)	1 (8%)	3 (25%)
Total		394 (42%)	261 (70%)	127 (34%)	191 (51%)

Sixteen of the 22 limbs were treated surgically and five aneurysms were not corrected. A variety of techniques was used for surgical corrections: 14 femoral popliteal bypasses were performed. In 10 of these, the aneurysm was ligated and in the remaining 4 it was excised. One interposition graft was performed after excision, one aneurysm was treated with ligation and iliopopliteal bypass because of an associated femoral aneurysm. One leg was amputated because of advanced gangrene due to a thrombosed popliteal aneurysm. The conduits used for bypass consisted of autogenous saphenous vein in eight, Goretex® in 4, modified umbilical vein in 3, and composite Goretex®-saphenous vein graft in 1.

Excellent results (normal ankle pulses) were obtained in ten (63 percent) cases and good results (asymptomatic limb) in five (35 percent) cases. One patient had a poor result due to thrombosis of the graft requiring amputation.

Complications consisted of graft thrombosis in two cases. One was reoperated with good results and the second was reoperated but eventually required amputation. Wound complications occurred in two and deep vein thrombosis and pneumonia each in one patient. Only one patient (6 percent) required amputation following reconstruction as described and he survived. The only mortality was related to the primary amputation in the patient with advanced gangrene who also was the oldest patient of this group (98 years).

COMMENTS

Popliteal aneurysms are not seen frequently and generally are undiagnosed until complications occur. The reason well may be the hidden location of this aneurysm in the popliteal space and lack of adequate examination of this part of the body during general physical examination. The complications often are reflected in an area distal to the anatomic site of the

TABLE 3

Associated Diseases

Author	No. of Patients	HTN	DM	CHD	CVA
Whitehouse[26]	61	23 (38%)	8 (13%)	20 (33%)	3 (5%)
Reilly[13]	159	62 (39%)	17 (11%)	52 (33%)	23 (14%)
Vermilion[12]	87	33 (38%)	15 (17%)	37 (42%)	3 (3%)
Szilagyi[2]	62	?	?	?	?
Guvendik[1]	21	6 (29%)	2 (10%)	7 (33%)	0 (0%)
Inahara[3]	30	10 (33%)	2 (7%)	8 (30%)	2 (7%)
Chitwood[18]	26	7 (27%)	?	8 (30%)	2 (8%)
Tompkins[16]	18	5 (28%)	2 (11%)	11 (61%)	4 (22%)
Alpert[27]	43	?	4 (9%)	?	?
Towne[17]	80	40 (50%)	12 (15%)	25 (31%)	10 (13%)
Hardy[28]	23	14 (61%)	?	?	?
Evans[29]	36	11 (31%)	2 (6%)	9 (25%)	0 (0%)
Wychulis[14]	152	102 (67%)	52 (34%)	67 (44%)	28 (18%)
Baird[6]	36	11 (31%)	3 (8%)	24 (67%)	8 (22%)
Edmunds[30]	82	48 (58%)	4 (5%)	26 (32%)	1 (14%)
Present Study	14	5 (36%)	3 (21%)	4 (28%)	0 (0%)
Total		377 (42%)	126 (14%)	298 (33%)	94 (11%)

aneurysm (digital or leg ischemia due to embolization or thrombosis) and this further masks the underlying problem from the casual observer. Complications are not related to the size of the popliteal aneurysm. Rupture and local compression of the popliteal vein or nerve are uncommon.

A review of the available literature on popliteal aneurysms is summarized in Tables 1 to 4. A total of 1,373 popliteal aneurysms in 930 patients is reported. Only 36 (3.8 percent) of the patients were female. Fifty percent of the cases were bilateral and 42 percent had associated extra popliteal aneurysms with 70 percent of those located in the abdominal aorta, 34 percent iliac, and 5 percent femoral.

Approximately one-third of the popliteal aneurysms were symptomatic when first detected and one-third were thrombosed. These figures are similar to our group.

Seventy percent of the aneurysms in these series were treated surgically, most commonly by autogenous saphenous vein or a synthetic graft bypass (74 percent). There was a slightly greater percentage of saphenous vein grafts as opposed to synthetic grafts used in the past ten years in comparison to the first ten years. Ten percent of extremities involved required amputation when first seen. Simple ligation or excision, with or without sympathectomy, and other forms of surgical therapy accounted for 14 percent of the procedures.

Popliteal aneurysms are infrequent with a ratio of approximately 1 popliteal aneurysm for every 15 or 16 abdominal aortic aneurysms,[1,2] but they are the most common form of peripheral aneurysms.[3] Aggressive surgical therapy is indicated since limb endangering complications are very common if they are untreated.[4] In a study of 100 of these aneurysms by Gifford et al. from 1913 to 1951, 49 cases presented with some form of complication and 10 additional complications developed. Thirteen additional complications later occurred in previously uncomplicated cases.[5] These figures justify an aggressive surgical approach.

Atherosclerosis is the etiology in the vast majority of cases, but other causes include penetrating wounds in the popliteal fossa,[6] syphilitic and mycotic popliteal aneurysms,[5] or operative injury, e.g. medial menisectomy.[7] False aneurysms of the popliteal artery due to trauma of a femoral osteochondroma have been described. With the advent of synthetic bypass grafts, pseudo or true aneurysms have been found at the site of the anastomosis with the native vessels both at the groin and popliteal sites.[9,10]

Continued enlargement of the popliteal aneurysm, despite proximal and distal ligation with bypass, has been described secondary to patent collateral arterial branches arising from the aneurysm.[11] This can be managed by ligation of these branches and obliteration of the aneurysm from within.

The clinical diagnosis usually can be made when one is evaluating symptoms of peripheral vascular insufficiency. Symptoms produced by popliteal aneurysms include calf claudication, rest pain, ulceration, and gangrene of the involved extremity.[12] Compression of neighboring venous and neural structures may cause venous occlusion or neurologic symptoms. Acute ischemia due to thrombosis, large embolus, or rupture may occur.[13] Rupture also may cause local compression symptoms in the popliteal space.[13] One feels a pulsatile mass if the aneurysm is patent, but thrombosis results in a nonpulsatile mass which may be confused as Baker's cyst or other popliteal mass.[14] Ultrasonography reliably diagnoses popliteal aneurysms, and digital subtraction angiography also is helpful.[15] Arteriography commonly is used to assess the run-off and collateral circulation and to plan the surgical procedure. Arteriogram may not reveal the presence of the aneurysm due to laminated clot in the lumen.[16]

The present treatment of choice for popliteal aneurysm is an aggressive surgical approach.[2-4,6,12,13,17,18] Revascularization in asymptomatic patients has better long-term results than when it is carried out in patients with symptoms or complications. The amputation rate is higher after revascularization in symptomatic patients.[14,17] The size of the popliteal aneurysm

TABLE 4

Treatment

Author	No. of Patients	Vein Graft	Prosthetic Graft	Ligation/ Excision
Whitehouse[26]	61	38 = (68%)	4 = (7%)	2 = (4%)
Reilly[13]	159	114 = (60%)	40 = (21%)	2 = (1%)
Vermilion[12]	87	50 = (41%)	49 = (40%)	0
Szilagyi[2]	62	30 = (60%)	20 = (40%)	0
Guvendik[1]	21	14 = (58%)	2 = (8%)	3 = (13%)
Inahara[3]	30	31 = (77%)	7 = (17%)	0
Chitwood[18]	26	24 = (70%)	5 = (15%)	1 = (3%)
Tompkins[16]	18	9 = (47%)	2 = (11%)	0
Alpert[27]	43	24 = (55%)	4 = (9%)	0
Towne[17]	80	9 = (10%)	62 = (70%)	4 = (5%)
Hardy[28]	23	?	?	?
Evans[29]	36	14 = (28%)	27 = (54%)	1 = (2%)
Wychulis[14]	152	21 = (27%)	23 = (29%)	10 = (13%)
Baird[6]	36	4 = (11%)	8 = (23%)	3 = (9%)
Edmunds[30]	82	31 = (32%)	34 = (35%)	0
Present Study	14	8 = (47%)	8 = (47%)	0
Total		421 = (44%)	295 = (31%)	26 = (3%)

Author	Amputation	Others	Surgical	Nonsurgical
Whitehouse[26]	4 = (7%)	8 = (14%)	56 = (67%)	32 = (36%)
Reilly[13]	23 = (12%)	12 = (6%)	191 = (78%)	53 = (22%)
Vermilion[12]	12 = (10%)	10 = (9%)	121 = (82%)	26 = (18%)
Szilagyi[2]	0	0	50 = (57%)	37 = (43%)
Guvendik[1]	4 = (17%)	1 = (4%)	24 = (86%)	4 = (14%)
Inahara[3]	0	2 = (5%)	40 = (91%)	4 = (9%)
Chitwood[18]	3 = (9%)	1 = (3%)	34 = (47%)	1 = (3%)
Tompkins[16]	6 = (33%)	2 = (11%)	19 = (73%)	7 = (27%)
Alpert[27]	4 = (9%)	12 = (27%)	44 = (69%)	20 = (31%)
Towne[17]	4 = (5%)	9 = (10%)	88 = (74%)	31 = (26%)
Hardy[28]	?	?	21 = (68%)	10 = (32%)
Evans[29]	6 = (12%)	2 = (4%)	50 = (89%)	6 = (11%)
Wychulis[14]	13 = (17%)	11 = (14%)	78 = (33%)	155 = (67%)
Baird[6]	10 = (28%)	10 = (28%)	35 = (69%)	16 = (31%)
Edmunds[30]	4 = (4%)	29 = (29%)	98 = (100%)	0
Present Study	1 = (6%)	0	17 = (77%)	5 = (23%)
Total	94 = (10%)	109 = (11%)	966 = (70%)	407 = (30%)

should not be a determining factor in deciding operative therapy, unlike abdominal aneurysms, since even small aneurysms may thrombose or cause other complications.[3]

Surgical treatment goes back to Galen who described a traumatic popliteal aneurysm.[18] Modern surgical therapy began in 1785 when Hunter ligated the femoral artery at mid-thigh in a 45-year-old coachman and obtained good results.[18] In 1902, Rudolph Matas performed an obliterative endoaneurysmorrhaphy for the treatment of a popliteal aneurysm.[19] This technique has been advocated as an alternative in poor-risk patients and in those with poor runoff.[20]

Autogenous vein grafts were first used in 1906;[18] in 1912, Hogarth Pringle in Britain[21] and, in 1916, Bertram Bernheim in the United States[22] used a saphenous vein in the repair of popliteal aneurysms. In 1935, Bird used sympathectomy and obliteration[23] and, in 1953, sympathectomy followed by excision of the aneurysm was recommended by Gifford.[5] With the introduction of synthetic grafts for arterial interposition in 1952,[24] a new era in vascular surgery began. By 1959, the usefulness of prosthetic grafts for the treatment of popliteal aneurysms was clearly established by Crawford, DeBakey, and Cooley.[25]

At present, we recommend bypass of the aneurysm with autogenous saphenous vein; if unavailable, a synthetic graft should be used with proximal and distal ligation of the aneurysm.[4,6,13,18] Excision of the aneurysm is not always necessary since damage to surrounding structures may occur.[26]

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INTUSSUSCEPTION OF SMALL BOWEL SECONDARY TO JEJUNAL DIVERTICULOSIS

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Symptoms, treatment, and complications of diverticulosis generally are well understood. Early diagnosis and appropriate medical and surgical treatment are essential to prevent high morbidity and mortality. A case of jejunal diverticulosis leading to intussusception secondary to aberrant location of one of the diverticula is presented.

Non-Meckelian small intestinal diverticula probably were described by Sommering in 1796.[1,2] The earliest records in English literature appear to have been made by Sir Astley Cooper in 1807, as he reported several autopsy cases.[3] In 1881, William Osler recognized multiple intestinal diverticula as a cause of intestinal obstruction.[4] The earliest operative description of intestinal obstruction secondary to small bowel diverticula was made by Cordinier and Sampson in 1906.[5] In 1920, Case demonstrated small bowel diverticula by barium x-ray examination.[6] In 1921, Hunt and Cook successfully excised a jejunal diverticulum.[7] Subsequent literature describes mainly a variety of complications associated with jejunal diverticulosis in previously asymptomatic patients.

CASE HISTORY

A 63-year-old white male carpenter was admitted to St. Francis Medical Center on June 22, 1984, with chief complaints of abdominal pain and vomiting which started one day prior to his admission. The pain which had increased in intensity was generalized, intermittent, and crampy in nature. Past medical history was insignificant except for a duodenal ulcer, treated medically since October 1982. Physical examination revealed a well-nourished male with moderate abdominal distention. The abdomen was diffusely tender with guarding and rebound and hyperactive bowel sounds were present. The remainder of the physical examination was noncontributory. Patient's temperature was 98.8; pulse 80; BP 150/190. Laboratory determinations revealed moderate leukocytosis with shift to the left (WBC 14.7; poly 75%; stab. 9%; lymph 8%; mono 8%) and hemoconcentration (Hgh 16.1; Hct 48.4). Amylase level was normal. Radiologic evaluation of the abdomen revealed no evidence of free air, but multiple air fluid levels were seen, as well as a few gas-filled nondilated loops of small and large bowel in the upper abdomen.

With the preoperative diagnosis of intestinal obstruction and generalized peritonitis, celiotomy was performed. The peritoneal cavity contained about 500 cc of turbid fluid. The stomach and duodenum were normal. The small bowel contained extensive diverticula with diameters varying between 1 to 3 cm (Figure 1), starting 1 foot below Treitz's ligament and extending approximately 2.5 feet distally in the proximal jejunum. These diverticula were located on the meso-side of the intestine with the exception of a larger one present on the antimesenteric border which measured 3-5-4 cm in its diameter (Figure 2). The adjacent jejunum had an edematous and erythematous segment, thickened and covered with fibrinous exudate that was believed to have been the site of intussusception with

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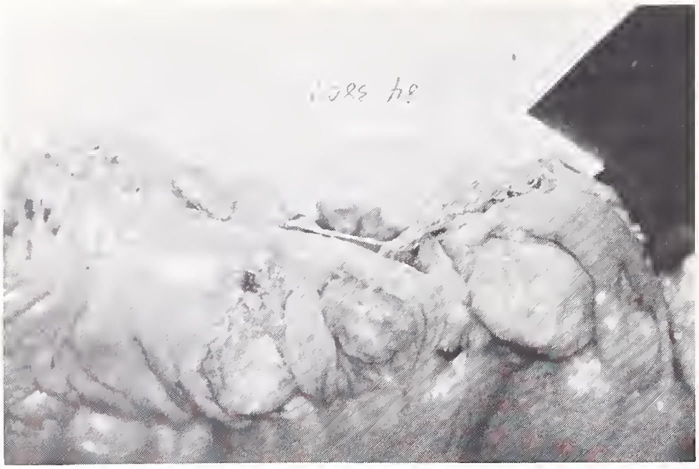

Figure 1—Multiple diverticula of the jejunum located on the mesenteric border.

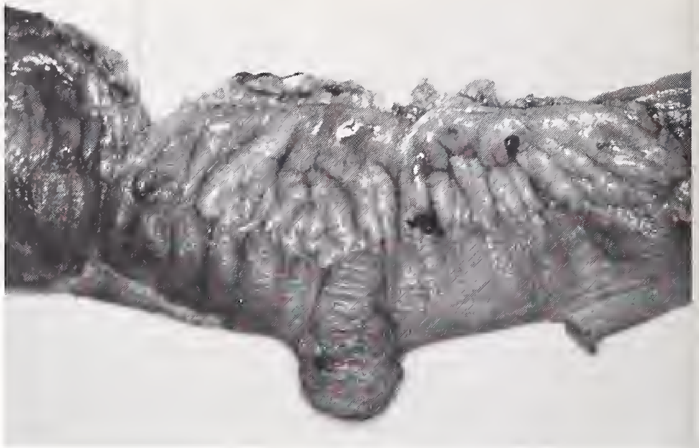

Figure 2—Large diverticulum located on the antimesenteric border of the jejunum.

the larger diverticulum as a leader. Although one may debate this impression, the presence of some heavy pasty material in the large diverticulum, consisting of vegetable fiber, and the absence of visible perforation of intestine made intussusception a reasonable possibility.

Exploration of the rest of his small intestine showed normal distal jejunum and ileum with the exception of a small Meckel's diverticulum in the usual location. Resection of the involved segment of the small bowel was performed with end-to-end anastomosis. Recovery was essentially unremarkable.

Pathological examination showed the serosal surface of the resected intestine to be markedly congested and covered by fibrinopurulent exudate. Upon opening the intestine, scattered erosive lesions of the mucosa were noted (Figure 3), as well as large amounts of undigested vegetable material packed tightly in the lumen. Microscopic examination revealed ischemic enteritis. In addition, examination of the large diverticulum present on the antimesenteric border revealed all layers of intestine to be present (Figures 4 and 5). This contrasts with the usual appearance of acquired diverticula. The peritoneal fluid was found to be an exudate and cultures were negative.

DISCUSSION

Although diverticulosis of the small bowel is regarded as relatively innocuous, recent studies suggest that the incidence and rate of complication may be increasing.[8] It is estimated that between 0.3 and 1.3 percent of the population harbors small intestinal diverticula. Edwards's survey of 2,820 autopsies noted an incidence of 0.3 percent.[9] Orr and Russell reviewed 2,161 barium contrast studies and found a prevalence of 0.42 percent.[10] The most accurate figures are from Cooke,[11] and Rosedale and Lawrence who reported an incidence of 1.25 percent and 1.3 percent, respectively.[12] Although reported in patients less than 20 years of age, small bowel diverticula frequently are seen in older strata of the population.[8] Their peak incidence is in the 6th and 7th decade.[8,13,14] They occur more frequently in males and are more prevalent in whites.[13]

PATHOGENESIS, CLINICAL PRESENTATION, AND COMPLICATIONS

The pathogenesis of jejunal diverticulosis is less well understood. The higher incidence in older patients,[8] with only occasional appearance on the antimesenteric border of the intestine,[10] and their association with colonic diverticula in almost 50 percent of cases, have led most authors to agree that non-Meckelian diverticula of the small intestine are acquired pulsion diverticula. Edwards, emphasizing this acquired nature, referred to the neurovascular entry site in the jejunal wall as the "locus minoris resistentia." He believed the relative weakness of the bowel wall at this site, along with the increased intestinal pressure secondary to irregular and active pristalsis, to be the cause of protrusion of the mucosa and submucosa through the mascularis of the intestine, resulting in formation of diverticula.

Jejunal diverticula appear as rounded or elliptical, single or multiloculated outpouchings of the intestinal wall. They rarely appear as a single, isolated structure, but usually are multiple, involving a continuous segment of intestine. Rarely, a cluster of diverticula can be seen separated by a variable length of intervening normal intestine. Diverticula occur on the mesenteric aspect of the bowel wall at the site of neurovascular penetration, but occasionally they will be seen on the antimesenteric portion of the bowel circumference. A branch of mesenteric vessel often can be seen coursing across the fundus of the sac. When numerous, the diverticula may vary in size measuring up to 10 cm in diameter.[13] The most frequent site of involvement is in the proximal jejunum. Since diverticula are formed by herniation of mucosa and submucosa through the muscular layer of the bowel, their wall consists of a thin covering layer of peritoneum lined by submucosa and mucosa. Although the smaller diverticula may present a muscular coat, the larger ones lack the well-developed, smooth muscle layer which is characteristic of the normal adjacent small intestine.[16] Another occasional finding is hypertrophy of the proximal jejunum with dilatation of the jejunal loops. This is thought to be due to resistance in the movement of the peristaltic waves and the contents of the intestinal lumen. Hence, in the affected intestine, peristaltic waves are very active but uncoordinated and inefficient, a state which has been termed as "jejunal dyskinesia."[13] Consequently, this produces stasis and resistance against the normal peristalsis, causing eventually hypertrophy of jejunal loops proximal to the diverticula.

Many individuals with jejunal diverticulosis remain

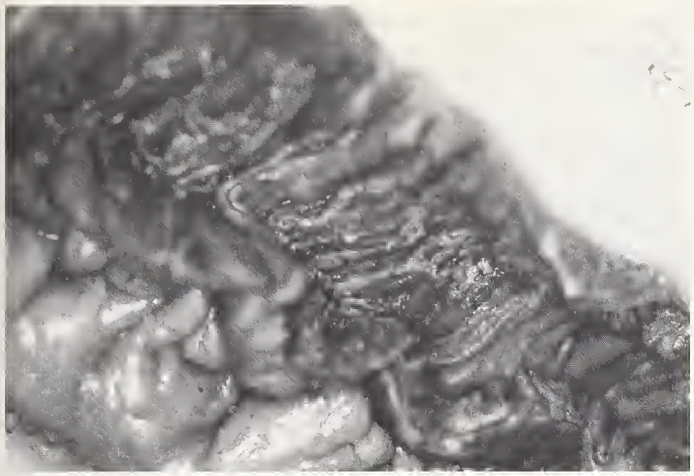

Figure 3—Ischemic enteritis changes in intussuscepted segment of jejunum.

asymptomatic, while the remaining manifest symptoms which mimic a variety of clinical entities. Presenting symptoms generally include subjective mild abdominal discomfort for which no cause can be found. Abdominal pain can be a prominent symptom; usually it is mild, generalized, intermittent, and related to food intake. The pain increases within an hour of meal ingestion. Some authors consider the pain as unrelated to food and attribute it to a low-grade inflammatory process around poorly emptying diverticula.[28] Other signs and symptoms, including anorexia, eructation, nausea, vomiting, flatus, diarrhea, constipation, and abdominal distention, often are attributed to other gastrointestinal or psychosomatic disorder. A few patients present with the more dramatic symptoms of severe complications including perforation, peritonitis, obstruction, or hemorrhage.

Acute diverticulitis, with or without perforation, appears to be the most common complication affecting diverticula of jejunum.[8,10,19] It is initiated by obstruction to free drainage of the diverticula with stasis and resulting excessive bacterial proliferation and tissue invasion;[20,21] this, coupled with raised intradiverticular pressure, leads to perforation.

In 80 percent of the cases reported by Herrington, perforation was attributed to an acute necrotizing inflammatory process.[15] In another 12 percent, abdominal trauma appeared to be the precipitating factor. The presence of a foreign body within the diverticulum seemed responsible for its rupture in another 6 percent of the reported series.

Hemorrhage should be regarded as a serious complication of jejunal diverticulosis, as emphasized by Shackleford and Marcus who reported 24 cases.[17] Bleeding may be massive or very slow presenting as chronic melena. Recurrent bleeding is reported to be common, as well.[22-24]

The exact mechanism of diverticular obstruction is not clear, but it is believed that the wide mouth of some diverticula and distention of other noncontracting diverticula may interfere with the effectiveness of intestinal peristalsis with consequent stasis and obstruction. In addition, existence of jejunal dyskinesia and resulting intestinal hypertrophy and dilatation occasionally produce a nonmechanical type of obstruction.[13] Furthermore, the unusual weight and volume of dilated intestinal loops affected by diverticula predisposes them to intermittent volvulus or intussuception.[26,29] Finally, true mechanical obstruction may occur from adhesions, developing at the site of repeated low-grade diverticular inflammation.

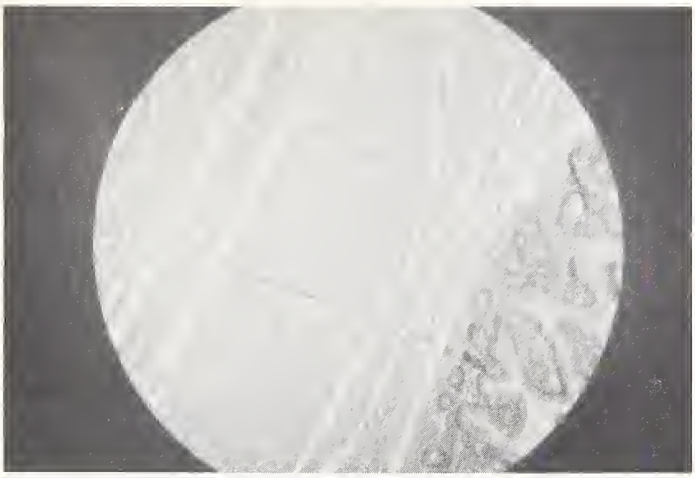

Figure 4—Small power view of section of jejunal antimesenteric diverticulum.

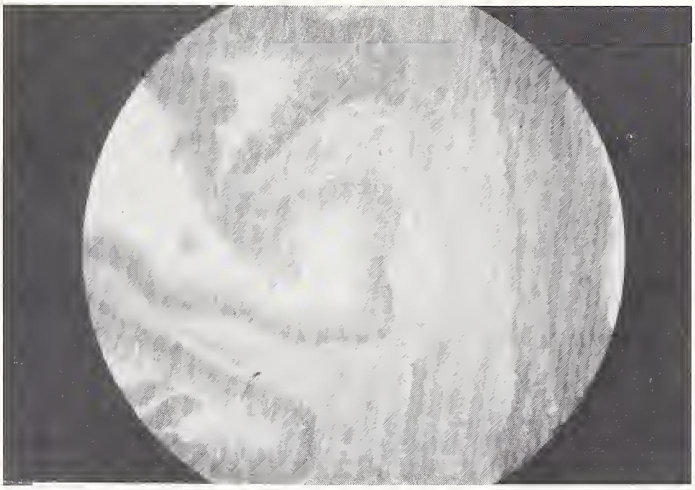

Figure 5—Higher magnification of antimesenteric small bowel diverticulum.

The metabolic abnormalities are the most interesting complication of jejunal diverticulosis. Knauser and Svoboda believed jejunal diverticulosis to be a physiologic equivalent of multiple "blind loops," in terms of production of malabsorption and related consequence.[18] After prolonged stasis, the jejunal lumen and diverticular outpouchings become colonized by bacteria, resulting in destruction of ingested nutrients and malabsorption, as well as production of toxic materials which, in turn, interfere with the normal absorption by the intestinal mucosa. Particularly, vitamin B12, fat, carbohydrate, and vitamin A are poorly absorbed in these patients.

Enterolith formation in a diverticulum of the jejunum is an uncommon complication. Haslewood and Roxburgh reported an instance where 12 brown facetted, lightly packed stones were seen in an inflamed diverticulum. Analysis showed that these stones were composed of bile acid, precipitated on vegetable materials.[25]

Other rare complications of jejunal diverticulosis include infestation with intestinal parasites[10] and traumatic rupture.[27]

TREATMENT AND PROGNOSIS

Treatment of jejunal diverticulosis varies with the manifestations of the disease. In patients with mild to moderate symptoms known to be due to jejunal diverticulosis, a period of medical treatment is warranted. The goal is to maintain good drainage of the jejunal diverticula. Therefore, a low residue diet may be very helpful. Antibiotics are indicated in cases of mild diverticulitis, but chronic disabling symptoms and acute complications require surgical treatment. Multiple surgical modalities are available depending upon the nature of the disease. Diverticulectomy with invagination of the stump is a satisfactory measure in the case of a solitary uninflamed lesion.[10] However, one has to bear in mind the possibility of intestinal lumen compromise should the diverticular base be wide. Resection with end-to-end anastomosis then is the method of choice. Complications of diverticular disease of the small bowel also are treated by resection and end-to-end anastomosis. Should diverticula involve a great length of small intestine, resection then should be limited to the proximal small bowel, since it harbors the largest diverticulum and has been shown to be the most common site of complications.

Diverticulosis of jejunum carries a high mortality rate. Munyaradzi and Wapnick reported 10 cases of multiple jejunal diverticulosis in 1973 with a mortality rate of 30 percent.[29] Herrington reported 21 percent mortality in his series of 33 cases.[15] In other series, mortality from perforated small bowel diverticula ranged between 21 to 40 percent.[16,23] This high mortality rate is related mostly to patients' age and to delays in diagnosis and treatment. Efforts at earlier diagnosis and appropriate surgical treatment remain the only means of reducing morbidity and mortality in these patients.

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PSYCHIATRIC DISORDERS PRESENTING AS NEUROLOGIC ILLNESS

KENNETH J. WEISS, M.D., CAMDEN*

Conversion disorder (hysteria), pseudoseizures, and pseudodementia are manifestations of psychiatric disorders that can mimic neurologic syndromes. The author provides clues to the differential diagnosis of these conditions.

It always is a diagnostic triumph when a psychiatrist can assert with assurance that the cause of a patient's behavioral, emotional, or cognitive impairment is a seizure disorder, tumor, stroke, or demyelinating disease. It is troublesome when a neurological disorder presents as a psychiatric illness, since a failed diagnosis can be fatal. This paper deals with the patient whose signs and symptoms indicate a neurologic illness, but the problem proves to be psychiatric. With the notable exception of depressive pseudodementia, which can lead to suicide, these conditions rarely have dire sequelae. These patients tax our diagnostic acumen and underscore the fact that, in the final analysis, clinical skills and intuition are our primary technology. The following discussion will focus on those disorders with most relevance to the practitioner, including conversion disorder, pseudoseizures, and pseudodementia.

CONVERSION DISORDER (CONVERSION HYSTERIA)

Conversion disorder, and the related somatization disorder, are classified with somatoform disorders in DSM-III.[1] There is not a great deal of grand hysteria these days, and probably not a great deal of advancement in its diagnosis and treatment since the days of Briquet, Charcot, Janet, and Freud.[2] The sorts of somatoform disorders that mimic neurologic illness are the classic hysterical reactions in females who show multiple and dramatic physical complaints with no apparent organic cause, and those in persons of either sex who become disabled in order to obtain monetary compensation or to avoid military duty or prison.[3] The symptoms of hysteria, which have much in common with anxiety disorders, are varied, and include: headache, blurred vision, lump in the throat, loss of voice, dyspnea, palpitations, anxiety attacks, anorexia, nausea and vomiting, abdominal pain, dysmenorrhea, sexual indifference, painful intercourse, paresthesias, dizzy spells, and emotional lability.[4] The clinician should consider the diagnosis of panic disorder when a patient presents with anxiety attacks and a disparate group of symptoms without an apparent precipitant.[5] Since panic disorder eminently is treatable, it would be regrettable for a patient to be unnecessarily relegated to the status of a hysteric or a hypochondriac—diagnoses with dubious prognoses.[6]

Conversion hysteria is rare and this fact alone should motivate us to use the full power of available diagnostic technology to rule out an organic etiology for any suspicious or atypical neurologic presentation.

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Occasionally, the symptoms of hysteria take the form of neurologic illness, and mimic paralyses, gait, and sensory disturbances or involuntary movements.

History and Physical Findings. Does the patient have a known anxiety disorder or somatoform disorder? The presence of a psychiatric disorder by history should not in itself distract us from the task of ruling out an organic disorder, such as complex partial epilepsy, stroke, multiple sclerosis, and other demyelinating diseases. Suggestibility should be looked for. The nonfixed nature of a motor or sensory defect is a reason to suspect hysteria. That is, when the symptoms can be modified by your making suggestions that the patient change, or when the symptoms vary from one presentation to another, a functional etiology should be suspected.

Pseudodementia, pseudoseizures, and conversion disorders are all examples of interface areas between psychiatry and neurology.

Conversion paralyses may affect any part of the body ordinarily under voluntary control.[3] Frequently, the movement across one joint or all or part of a limb may be involved. The face and tongue infrequently are involved in conversion. The distribution of the paralysis corresponds to the patient's idea of anatomy, so that a paralysis across a joint is never organic. When the hysterical patient attempts to move the affected part, he/she contracts both the primary muscles and their antagonists. For example, a patient asked to flex the leg also will contract the quadriceps. Wasting and contractures are extremely rare and the electromyogram (EMG) is normal in conversion disorder.

Deep tendon reflexes if not precluded by rigidity are symmetrical and not diminished.[3] Plantar reflexes are flexor. Muscular rigidity may be localized to an affected limb or generalized, as in a hysterical trance. In hysteria, rigidity increases in proportion to the effort made to move the limb. Coarse tremors are seen frequently. They increase when given attention and may disappear when the patient's attention is distracted.

Like the motor examination, the sensory findings often are anomalous in their anatomical distribution. Typically, there are sharp upper borders of anesthesia or a pattern that clearly does not follow the distribution of the spinal nerves. Coordination may be perfect despite an apparent loss of position sense. Hysterical deafness can be shown to be functional when the patient can be awakened from sleep by a noise. In hysterical blindness, the optic discs and pupillary reactions are intact and the patient may be seen to avoid obstacles.

The gait typically is bizarre and does not resemble an organically disturbed gait. There is a tendency for the patient to fall and not get hurt. There may even be difficulty standing; hysterical patients are more difficult to support than neurologic ones who at least make an effort. If one leg is affected, one does not see typical circumduction, but rather the patient pushes or drags the leg. Adams and Victor describe a hysterical gait: "Astasia-abasia, in which patients, though unable to either walk or stand, retain normal use of their legs in bed, nearly always is hysterical. When such patients are placed on the floor they take a few normal steps and then become unable to advance their feet; they lurch wildly and crumple to the floor if unassisted." The authors caution, however, that there are neurologic conditions that must be ruled out including lesions of the frontal lobe and anterosuperior cerebellar vermis.[4]

PSEUDOSEIZURES

Seizure-like behavior is much more likely to be encountered than grand hysteria. All of the major seizure disorders can be mimicked by patients with anxiety, somatoform, and personality disorders. To our chagrin, even patients with otherwise bona fide seizure disorders can show pseudoseizure activity as part of an attention-seeking behavior pattern. Psychiatrists constantly look for ways to redefine clinical presentations of bizarre episodic behavior as manifestations of complex partial epilepsy, since it has clear treatment implications.[8] In my experience, EEG studies are a disappointingly insensitive screening test for patients showing episodic behavioral problems, but some authors recommend long-term EEG studies.[9] Trials with anticonvulsant drugs are similarly not suited to the usual patient with a personality disorder or somatization, but we should not abandon such efforts, as the successful interventions are dramatic and rewarding.[10]

Leicester reported the difficulties in tying together behavior and epilepsy.[11] He reviewed 17 patients referred by psychiatrists to neurologists as adults with temper tantrums. All had normal EEGs and identifiable psychological precipitants. The author cites the widely held misbelief that complex partial epilepsy presents with temper tantrums or random violence. Interictal aggressiveness is well known, but purposeful violent behavior during an ictus is less common. Rage-like attacks can occur as a result of such organic conditions as tumors of the hypothalamus, cingulum, septum, or third ventricle.

The opposite experience was reported by Lewis et al. who examined 97 incarcerated delinquent boys.[12] They found 18 boys to have psychomotor epileptic signs and symptoms although not all of them had positive EEGs. The degree of violence correlated highly with the amount of psychomotor signs and several of the boys had clear ictal as well as interictal violence.

Episodic dyscontrol, a disorder of controversial status, straddles the border of psychiatry and neurology.[10] These patients show explosions of rage in unpredictable seizure-like ways. Although these patients may not fulfill formal criteria for epilepsy, they may be one of the best examples of what one might call an organic personality disorder.[13] Clinicians are more or less obligated to treat these patients with a trial of anticonvulsants, since the prospects for interpersonal treatment of their disorder are dismal.

The biological substrate that leads a patient to "choose" pseudoseizures as an expression of emotional distress is a frontier area for study. It is not difficult to understand the secondary gains of a true epileptic involved in attention-seeking or angry outbursts, using learned behaviors to elicit help from others. This situation can be particularly frustrating to the clinician who then must learn to "read" different kinds of seizure-like behavior.

Some physical aspects of patients with suspected pseudoseizures can aid in differential diagnosis. Hyperventilation frequently is seen in anxious and phobic patients. When severe, this sign can lead to loss of consciousness or tetany, which can mimic seizure activity. Severe episodic hyperventilation, phobias, and anxiety should lead one to entertain a diagnosis of panic disorder. This is a particularly timely diagnosis, since panic disorder is treatable with antidepressant medications.[6] Hysterical pseudoseizures or malingering (intentional symptom simulation) typically lack an aura, initiating cry, hurtful falls, and incontinence. There may be peculiar movements, such as grimacing, biting, squirming and resisting assistance, and striking out at persons trying to assist. Consciousness is retained during what appears to be a bilateral major motor seizure. The pseudoseizure activity frequently can be terminated by strong noxious stimulation and memory for the event can be elicited through hypnosis or drug interview. After ruling out epilepsy, the appropriate action plan is structured psychotherapy in a day treatment center where the immediate antecedents of the behavior can be identified and worked into the therapy. It is not uncommon for pseudoseizures to disappear rapidly in a supportive environment. In the case of borderline personality disorder, the behavior most often will reappear transiently during times of stress, the therapist's vacation, and anniversary dates of losses.

PSEUDODEMENTIA

In pseudodementia, the signs and symptoms of dementia are mimicked or caricatured by functional psychiatric illness.[14] Depression-induced dementia may be indistinguishable from organic mental disorder.[15] It has been estimated that 10 to 15 percent of our nation's elderly have some degree of dementia, but that 10 percent of these persons actually have depression as the primary illness.[16] Depression in the elderly is very significant; it is the most notable example of a neurological presentation of a psychiatric disorder which is potentially fatal, as suicide is not uncommon in this group. The patient with unrecognized depressive pseudodementia is at risk for both therapeutic neglect and unnecessary exposure to invasive diagnostic procedures.[17]

Depression must be differentiated from true dementia, and from pharmacologically, metabolically, or neurologically induced delirium. One of the major obstacles to the correct diagnosis of depression is the widely held misbelief that persons naturally become demented as they grow older. Family members and physicians fall prey to therapeutic nihilism and many patients with reversible depressions, dementias, and deliria are relegated to supportive care settings. A second problem in recognition of depression is that elderly depressed patients may have a different clinical presentation than younger ones.[16] Symptoms in the elderly often include anergia, memory loss, decreased concentration, psychomotor retardation, social withdrawal, irritability, vegetative signs, and various somatic complaints. Some less prominent symptoms may be experienced—sadness or crying and a diminished capacity to directly express guilt and suicidal ideas.

There are many reversible conditions with medical or iatrogenic causes that can contribute to dementia, delirium, and depression.[16] As a rule of thumb, any abrupt onset and short duration mood change is depression and not dementia, although both may have an insidious onset. Medical diseases associated with depression include diabetes, hypoglycemia, hyper- and hypothyroidism, infections, parkinsonism and other central nervous system lesions, and many chronic diseases. The pharmacologic causes for depression also are numerous and include various antihypertensives, most psychotropic agents, antiparkinson drugs, digitalis, and estrogens. Since the iatrogenic causes of depression-induced dementia or drug-induced delirium are significant and reversible, patients being evaluated for this disorder should have the benefit of blood level monitoring of all appropriate drugs. This is an elegant and essential step in the workup, especially since a finding of toxicity at this early stage spares the patient from lengthy and invasive diagnostic procedures.

DIAGNOSIS OF DEPRESSION-INDUCED (PSEUDO-) DEMENTIA

1. Obtain an accurate history of the illness, including time course of onset, premorbid personality, any daily fluctuations in functioning, medical problems, and recent drug ingestions.
2. Is there a history of depressive illness in relatives? Has the patient or any relative been successfully treated with an antidepressant, lithium, or electroconvulsive therapy?
3. Laboratory studies should include standard tests, such as CBC, urinalysis, vitamin B-12 and folate blood levels, thyroid studies, and a CT scan of the head.
4. A drug screen or, preferably, quantitative blood levels of any measurable drugs should be obtained.
5. The Dexamethasone Suppression Test (DST) is highly specific, quite sensitive, and positive in at least 50 percent of cases of severe depression. When positive (i.e. nonsuppression of plasma cortisol), it can discriminate between depression and dementia.[16] However, there are reports that nondepressed demented patients frequently are DST-positive.[18,19]
6. When the electroencephalogram (EEG) is diffusely abnormal, i.e. consistent with dementia or delirium, one often can rule out depression. However, since depressions also are quite common in patients with organic mental disorders, a positive EEG does not in itself rule out the possibility that the depression is contributing substantially to the cognitive dysfunction.
7. Routine cognitive testing or specialized neuropsychological testing may be helpful in defining the limits of apparent cognitive dysfunction. Testing especially is useful when there is a discrepancy between behavior and objective performance, which usually is a sign of depression.
8. The barbiturate interview is used to discriminate between functional and organic states, e.g. in cases of dementia, amnesia, and catatonia.[20] The functionally

ill patient will show a temporary clearing, whereas the organic patient will get worse, will sleep, or become delirious.

Wells has summarized the clinical findings that help differentiate dementia and depression-induced pseudodementia.[14] Primary dementia tends to have a gradual onset and a slow progression, whereas depression is typically of shorter duration before medical help is sought. The demented patient usually is oblivious to the extent of the cognitive loss, complains little, and tends to rely on adaptations such as keeping notes and calendars. This patient will try hard to perform, shows a shallow and labile affect, and demonstrates behavior congruent with the severity of the cognitive disturbance. The pseudodepressed patient complains a great deal even though his behavior is not congruent with the true level of cognitive loss. This patient will have a more pervasive affective change and will not try to maintain a performance level, preferring to dwell on failures. On testing of intellect, the demented patient will have faulty attention and concentration, try to answer but give near-misses, and score consistently on tasks of similar difficulty. Here, the depressed patient will give "I don't know" answers, despite good attention and concentration and show variability in tasks of similar difficulty. These differences are most marked in patients with depressive or dependent temperaments or histrionic personality disorders.[15]

The treatment for pseudodementia depends upon the determined etiology. Inasmuch as depression is a treatable disorder and primary degenerative dementia is not, it is in the interest of the patient to place emphasis on the diagnosis of depression. In equivocal cases, treatment with an antidepressant is justified. Frequently there will be a lifting of mood, leaving a residual organic impairment of less overall severity. For those depressed patients for whom antidepressants have failed, or who are in danger of death, unilateral electroconvulsive therapy (ECT) becomes the treatment of choice.[16] Special care must be taken not to exacerbate any concurrent primary dementia.

CONCLUSION

As the neural substrates of behavior are elucidated, fewer disorders can be classified as "functional." Nevertheless, there are interface areas between psychiatry and neurology that call upon specialized diagnostic and clinical interview procedures. Pseudodementia, pseudoseizures, and conversion disorder are all examples of this area. We have seen that careful attention to irregularities in the patients' presentations helps to formulate a meaningful differential diagnosis.

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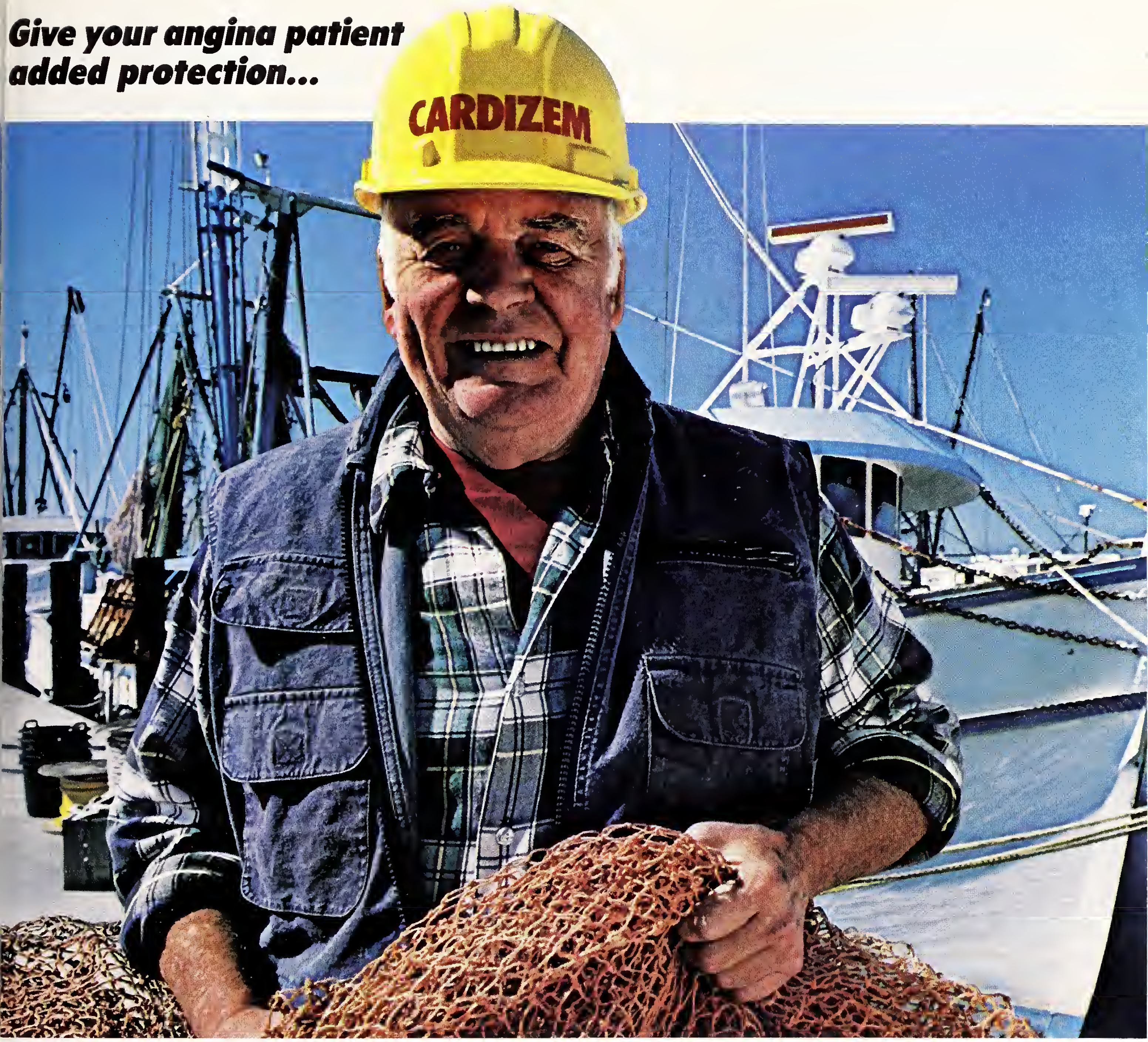

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There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction**. CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure**. Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM (diltiazem hydrochloride) in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered.

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy** — CARDIZEM may be safely co-administered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers**. (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

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References: 1. *Physicians' Desk Reference®*, ed 39. Dradell, NJ, Medical Economics Company Inc, 1985. **2.** Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): *Heart Disease: A Textbook of Cardiovascular Medicine*, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39. **3.** Schroeder JS: Calcium and beta blockers in ischemic heart disease. When to use which. *Mod Med* 1982; 50(Sept):94-116. **4.** Subramanian VB: Comparative evaluation of four calcium antagonists and propranolol with placebo in patients with chronic stable angina. *Cardiovasc Rev Rep* 1984; 5:91-104. **5.** Schroeder JS, Feldman RL, Giles TO, et al: Multiclinic controlled trial of diltiazem for Prinzmetal's angina. *Am J Med* 1982; 72:227-231. **6.** Weiner DA, McCabe CH, Cutler SS, et al: The efficacy and safety of high-dose verapamil and diltiazem in the long-term treatment of stable exertional angina. *Clin Cardiol* 1985; 7:648-653. **7.** Shapiro W: Calcium channel blockers: Actions on the heart and uses in ischemic heart disease. *Consultant* 1984; 24(Dec):150-159.

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DOCTORS' NOTEBOOK

Trustees' Minutes; UMDNJ Notes; AMNJ Report; AIDS Hotline; Physicians Seeking Location in New Jersey

Trustees' Minutes
March 16, 1986

A regular meeting of the Board of Trustees was held on Sunday, March 16, 1986, at the Executive Offices in Lawrenceville. Detailed minutes are on file with the secretary of your county society. A summary of significant actions follows:

Report of the President . . .
(1) Annual Meeting of the Medical Society of the State of New York (MSSNY) . . .
(a) Preadmission Certification Guidelines . . . Noted Dr. Fioretti's request to adopt the guidelines of the MSSNY concerning preadmission certification.
(b) Statewide IPA/HMO . . . Noted that a committee will be formed to begin preliminary investigation of the feasibility of establishing an IPA/HMO in New Jersey. Noted that this committee is comprised of Board members: Drs. Carnes, Gingerelli, Hirsch, Palace, Robins, Schauer.
(2) Edward J. Ill Award Recipient . . . Noted that Palma E. Formica, M.D., will receive the prestigious Ill Award from the Academy of Medicine of New Jersey.

Report of Executive Director . . . MSNJ Membership . . . Noted that total paid membership on February 28, 1986, was 5,714.

NJ Hospital Association . . . Was advised by Mr. Scibetta that the NJHA is trying to address the information reported in the media concerning mortality rates of hospitals.

Council on Public Health . . . Approved the following recommendation:

> That the Medical Society of New Jersey support public water fluoridation as a clearly demonstrated, safe, and effective way to prevent tooth decay.

Old Business . . .
(1) Petition for House of Delegates Seating . . . Rejected the Society of Surgeons of New Jersey petition for House of Delegates seating because they failed to meet the established criteria.
(2) Ad Hoc Committee on Health Care Reimbursement Policies . . . Voted to postpone the recommendation of the Committee for the issuance of a periodical on the socioeconomic issues facing physicians.

New Business . . .
(1) Injections Administered by Allied Medical Personnel . . . Voted to submit to the 1986 House of Delegates a resolution seeking passage of regulations or legislation to authorize formally trained allied medical personnel, in addition to R.N.s, be allowed to administer subcutaneous injections and perform skin tests under the immediate supervision of a physician.
(2) Radiologic Technology Board of Examiners . . . Requested possible candidates for the Radiologic Technology Board of Examiners to replace the late Dr. Goracci.
(3) Physicians' Union . . . Noted that Mr. Maressa will speak at a meeting of Mercer County physicians concerning physicians' unions. Noted that Mr. Maressa believes the National Labor Relations Board is unlikely to grant unions accreditation.

UMDNJ Notes

Stanley S. Bergen, Jr., M.D.
President

When the College of Medicine and Dentistry of New Jersey was founded in 1970 by act of the state legislature, one of its missions was to consolidate and unify all of New Jersey's public programs in medical and dental education.

In 1981, CMDNJ became a university—UMDNJ—in recognition of its growth and for providing a statewide system for health. Spreading health, education, and research to the residents of New Jersey's 21 counties have been formidable, yet achievable, tasks, due largely to the success of the affiliation agreements UMDNJ has with 100 hospitals and other health care educational and social service agencies that are involved in university teaching programs.

With continued success in mind, we have entered into a major affiliation agreement with Saint Barnabas Medical Center in Livingston; it promises to expand and enhance health care services not only in Essex County, but around the state as well, and to create new opportunities in medical education for medical students and physicians. This new partnership involves the development of programs in patient care, medical education, and research between the UMDNJ-New Jersey Medical School and Saint Barnabas.

We particularly are encouraged with this new affiliation because the 705-bed Saint Barnabas Medical Center is a renowned facility whose commitment and service to patients spans more than 120 years. Founded in Newark in 1865, Saint Barnabas moved to its present quarters in 1964. It is the largest nonprofit hospital in the state, and had more than 30,000 patient admissions and approximately 24,000 emergency-room patients last year. It also houses the only certified burn unit in the state.

Although the affiliation is in the planning stages, UMDNJ students already are involved in clerkships at the Livingston-based institution under the tutelage of Saint Barnabas physicians who have teaching appointments at the University.

The two institutions also are developing joint postgraduate programs for physicians in specialty training and for programs in continuing medical education for practicing physicians. These include plans to establish a collaborative open-heart surgery program at

Saint Barnabas and a lithotripsy service at University Hospital, which will be available to all New Jersey residents. The lithotripsy service is just one of many opportunities we have to introduce state-of-the-art medical technology in a cost-efficient manner.

The affiliation underscores UMDNJ's determination to reach out to hospitals around the state and work jointly on education, research, and patient care initiatives, and health care delivery. I am certain the benefits will be invaluable for all concerned. Physicians, students, and patients have much to gain from this joining of professional talent, medical education, resources, and services.

AMNJ Report

Dennis R. Filippone, M.D.
President

The Board of Trustees of the Academy of Medicine of New Jersey has named the 1986 recipients of the awards which will be presented at our Annual Awards Dinner on Wednesday, May 28, 1986, to be held at the Chanticler in Short Hills.

Palma E. Formica, M.D., will receive the Edward J. Ill Award "presented annually to that physician of New Jersey who merits recognition by the Academy for distinguished service as a leader in the medical profession and in the community at large." Recent recipients have included Drs. Arthur Bernstein, Victor Parsonnet, James Todd, Leon Smith, Arthur Krosnick, Alfred Alessi, and Stanley Bergen. Dr. Formica is best known to us as an officer, AMA Delegate, and past Board Chairman of MSNJ; however, she has been an outstanding leader in many other areas of medicine and the community.

The Academy's Citizen's Award is "presented annually to that citizen or group of citizens of New Jersey who merit recognition by the Academy for distinguished service in the interest of the health and welfare of the community at large." The 1986 Citizen's Award has been granted to Mr. Malcolm A. Borg. Mr. Borg is Chairman of the Board of Macromedia Incorporated which, among other interests, publishes *The Record* and *The Sunday Record*, New Jersey's second largest newspaper. Mr. Borg's contributions to the health care scene in New Jersey are numerous and include chairmanship of the Health Care Administration Board, membership on the Blue Ribbon Committee on Corporate Restructuring of New Jersey Hospitals of the State Health Coordinating Council, and most recently, the Governor's Advisory Committee on Capital Expenditures for Health Care Facilities.

The annual substance abuse symposium cosponsored with MSNJ was held on Wednesday, April 16, 1986, at the headquarters building in Lawrenceville. The topic for the all-day meeting this year was AIDS and the IV drug abuser and the program chairman for the meeting was Dr. Daniel Greenfield.

The Academy continues to pursue efforts toward a computer bulletin board. This service is expected to commence within the next few months. The service will include listing of scheduled AMNJ programs and an open format for leaving messages. Several computer software companies also have expressed interest in placing demonstration packages on-line whereby AMNJ Fellows could "down load" them for trial before contacting the vendor for a complete demonstration. Software might include CME Packages, Practice Management Programs, and Diagnostic Programs.

The speaker for the Academy's First Wednesday program for April was Mr. Peter Sweetland, President, Medical Inter-Insurance Exchange of New Jersey. Mr. Sweetland address the malpractice issue—a national perspective (including a discussion of how rates are calculated differently by specialty).

AIDS Information Hotline

The Medical Society of New Jersey in cooperation with the Academy of Medicine of New Jersey's AIDS Roving Symposium has prepared a listing of physicians who are knowledgeable and experienced in the diagnosis and treatment of the AIDS patient.

These physicians have volunteered to be a resource to physicians who have questions regarding AIDS, particularly as it relates to the rendering of care to the AIDS patient.

Spartaco Bellamo, M.D.
330 Clinton St.
Hoboken, NJ 07030
(201) 656-7824

Roger Cooper, M.D.
St. Michael's Medical Center
268 Dr. Martin Luther King, Jr. Blvd.
Newark, NJ 07102
(201) 877-5000

Richard Dixon, M.D.
Helene Fuld Medical Center
750 Brunswick Ave.
P.O. Box 2428
Trenton, NJ 08638
(609) 394-6031

William Farrer, M.D.
St. Elizabeth's Hospital
225 Williamson St.
Elizabeth, NJ 07207
(201) 527-5010

Isabel Guerro, M.D.
20 Apple Way
Marlton, NJ 08053
(609) 723-4221 ex. 228

Howard Holtz, M.D.
UMDNJ-NJ Medical School
100 Bergen St.
Newark, NJ 07103
(201) 456-4952

Edward Johnson, M.D.
St. Michael's Medical Center
268 Dr. Martin Luther King, Jr. Blvd.
Newark, NJ 07102
(201) 877-5000

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Rajendra Kapila, M.D.
185 Central Ave.
East Orange, NJ 07018
(201) 456-4802

Donald Louria, M.D.
UMDNJ-NJ Medical School
100 Bergen St.
Newark, NJ 07103
(201) 456-4422

Stephen Manocchio, M.D.
St. Mary's Hospital
308 Willow Ave.
Hoboken, NJ 07030
(201) 656-7824

Dominic Mauriello, M.D.
75 Bentley Ave.
Jersey City, NJ 07306
(201) 332-6387

Mary Ann Michelis, M.D.
Hackensack Medical Center
30 Prospect Ave.
Hackensack, NJ 07601
(201) 441-2065

John Middleton, M.D.
Old Bridge Regional Hospital
Division of Raritan Bay Medical Center
1 Hospital Plaza
Old Bridge, NJ 08857
(201) 442-3700

Anthony Minnefor, M.D.
St. Joseph's Hospital and Medical Center
703 Main St.
Paterson, NJ 07503
(201) 977-2181

Jeremias Murillo, M.D.
Newark Beth Israel Medical Center
201 Lyons Ave.
Newark, NJ 07112
(201) 926-7328

James Oleske, M.D.
UMDNJ-NJ Medical School
100 Bergen St.
Newark, NJ 07103
(201) 456-5066

Robert Palinkas, M.D.
UMDNJ-NJ Medical School
100 Bergen St.
Newark, NJ 07103
(201) 456-4300

George Perez, M.D.
St. Michael's Medical Center
268 Dr. Martin Luther King, Jr. Blvd.
Newark, NJ 07102
(201) 877-5000

Richard Porwancher, M.D.
St. Francis Medical Center
601 Hamilton Ave.
Trenton, NJ 08629
(609) 599-5000

M. Christine Reyelt, M.D.
St. Joseph's Hospital and Medical Center
703 Main St.
Paterson, NJ 07503
(201) 977-2000

John Salaki, M.D.
60 Franklin St.
Morristown, NJ 07960
(201) 540-8484

Pernendu Sen, M.D.
Jersey Shore Medical Center
1945 Corlies Ave.
Neptune, NJ 07753
(201) 775-5500

John Sensakovic, M.D.
St. Michael's Medical Center
268 Dr. Martin Luther King, Jr. Blvd.
Newark, NJ 07102
(201) 877-5487

Leon Smith, M.D.
St. Michael's Medical Center
268 Dr. Martin Luther King, Jr. Blvd.
Newark, NJ 07102
(201) 877-5000

Flor Tecson-Tumang, M.D.
Veterans Administration Medical Center
South Center and Tremont Ave.
East Orange, NJ 07019
(201) 676-1000 ex. 532

Jules Titelbaum, M.D.
Newark Beth Israel Medical Center
201 Lyons Ave.
Newark, NJ 07112
(201) 926-7328

New Jersey Department of Health AIDS HOTLINE
1-800-624-2377
Mon.-Fri. 8:45 A.M.-4:30 P.M.
(609) 588-3520
(201) 266-1910

New Jersey AIDS HELPLINE
Mon.-Fri. 7:00 P.M.-11:00 P.M.
(201) 596-0767

Physicians Seeking Location in New Jersey

The following physicians have written to the Executive Offices of MSNJ seeking information on possible opportunities for practice in New Jersey. The information listed below has been supplied by the physician. If you are interested in any further information concerning these physicians, we suggest you make inquiries directly to them.

ANESTHESIOLOGY—Julio Araoz, M.D., 4 Avon Way, Parlin, NJ 08859. San Andres (Bolivia) 1959. Board eligible. Any type practice. Available.

Yellagondahalli V. Janardhan, M.D., 18 Agatha Dr., Edison, NJ 08817. Bangalore (India) 1971. Board certified. Group, partnership, fee for service. Available.

Ashokkumar J. Patel, M.D., 500 Central Ave., Apt. 1113, Union City, NJ 07087. M.P. Shah (India) 1977. Board eligible. Group, partnership, solo, hospital based. Available.

CARDIOLOGY—Mian Arshad Jan, M.D., 6901 Old York Rd., Apt. B-208, Philadelphia, PA 19126. Khyber Medical (Pakistan) 1974. Also, internal medicine. Board certified (IM). Board eligible. Group or partnership. Available July 1986.

EMERGENCY MEDICINE—Julian A. Sellek, M.D., 1501 S. Greenway, Coral Gables, FL 33134. Salamanca (Spain) 1963. Also, family practice. Board eligible. Board certified (FP). Group, solo, partnership. Available.

ENDOCRINOLOGY—Michael J. Miller, M.D., 2375 Hudson Ter., Apt. 3-E, Fort Lee, NJ 07024. SUNY-Downstate 1981. Also, internal medicine. Board certified (IM). Board eligible. Group or partnership. Available July 1986.

FAMILY MEDICINE—James Ditaranto, D.O., 3901 Manayunk Ave., Philadelphia, PA 19128. Osteopathic (Philadelphia) 1984. Board eligible. Group or partnership. Available.

Lawrence S. Gordon, M.D., 1016 Spinnaker Way, Apt. G-1, Melbourne, FL 32935. Rutgers 1982. Board certified. Group, partnership, solo. Available.

Natalia Moishezon, M.D., 207 Christie St., Leonia, NJ 07605. Moscow 1964. Board eligible. Group, partnership, industry. Available.

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Jeffrey C. Schultz, M.D., 301 Victoria Dr., Monroeville, PA 15146. University of Massachusetts 1983. Board eligible. Group. Available July 1986.

Julian A. Sellek, M.D., 1501 S. Greenway, Coral Gables, FL 33134. Salamanca (Spain) 1963. Also, emergency medicine. Board certified. Board eligible (EMER). Group, solo, partnership. Available.

Lew Herbert Valliant, M.D., 115 Mountain View Dr., Elkins, WV 26241. Queen's University (Canada) 1976. Board certified. Solo or group. Available.

GASTROENTEROLOGY—Richard J. Fastiggi, M.D., 10 Silver Stirrup Ct., Timonium, MD 21093. UMDNJ 1977. Board certified (IM). Solo, partnership, group. Available July 1986.

Prasad Satyanarayana Maturu, M.D., 8863 17th Ave., Brooklyn, NY 11214. Mysore Medical (India) 1977. Board eligible. Group or partnership. Available June 1986.

Brian C. Weiner, M.D., 24 Bowling Green Pl., Staten Island, NY 10314. SUNY-Downstate 1981. Board certified. Group or partnership. Available July 1986.

INTERNAL MEDICINE—Walter M. Bakun, M.D., 199 Casterline Rd., Denville, NJ 07834. St. George's University (Grenada) 1983. Board eligible. Group, partnership, solo. Available July 1986.

Joseph Cohen, M.D., 16 Mountainbrow Village, Corning, NY 14830. New York Medical College 1982. Board eligible. Group. Available July 1986.

Arkady S. Fishman, M.D., 55 Nagle Ave., Apt. 1E, New York, NY 10040. Aktyubinsk Medical Institute 1969. Board certified. House staff, group, nursing home. Available.

Eli R. Goldner, M.D., 2841 W. Rosemont, Chicago, IL 60659. Rome (Italy) 1980. Board eligible. Group, HMO, partnership. Available.

Arpad C. Heinrich, M.D., 360 W. Pleasantview Ave., Hackensack, NJ 07601. Temeschwar (Transylvania) 1980. Board eligible. Solo, partnership, group. Available June 1986.

H. Homayouni, M.D., 459 Hamilton Pl., Hackensack, NJ 07601. Tehran (Iran) 1971. Board certified. Partnership. Available.

Mian Arshad Jan, M.D., 6901 Old York Rd., Apt. B-208, Philadelphia, PA 19126. Khyber Medical (Pakistan) 1974. Also, cardiology. Board certified. Board eligible (CARD). Group or partnership. Available July 1986.

Serge I. Kaftal, M.D., 125 Gates Ave., Apt. 38, Montclair, NJ 07042. Lodz Medical Academy (Poland) 1978. Solo, group, health center service. Available.

Rey Chi Lin, M.D., 714 Indian Creek Dr., Wilkes-Barre, PA 18702. China Medical 1977. Board eligible. Group, partnership, solo. Available July 1986.

Gerald B. Miele, M.D., 293 Demott St., Mineola, NY 11501. Rush 1983. Board eligible. Solo or partnership. Available July 1986.

Michael J. Miller, M.D., 2375 Hudson Ter., Apt. 3-E, Fort Lee, NJ 07024. SUNY-Downstate 1981. Also endocrinology. Board certified. Board eligible (ENDOCRIN), Group or partnership. Available July 1986.

Vijay Mittal, M.D., 35-44—28th St., Apt. 5-C, Long Island City, NY 11106. All-India 1978. Board eligible. Group or solo (part-time). Available July 1986.

Makkena Ramachandra Rao, M.D., 501 Indian Creek Dr., Wilkes-Barre, PA 18702. Guntur Medical (India) 1974. Board eligible. Group or partnership. Available July 1986.

Danuta Silber, M.D., 340 West End Ave., NY 10023. UMDNJ (PGY-3), Poland 1979. Board eligible. Group or partnership. Available July 1986.

Marian Lynn Taylor, M.D., 7 Loriann Rd., Warren, NJ 07060. UMDNJ 1983. Board eligible. Group or partnership. Available June 1986.

Dipak Vora, M.D., 509 Indian Creek Dr., Wilkes-Barre, PA 18702. University of Bombay (India). Board eligible. Available July 1986.

The *Journal* of the Medical Society of New Jersey
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NEUROLOGY—Pandurang R. Miskin, M.D., 5315 Minnehaha Ave., #105, Minneapolis, MN 55417. Minnesota 1986. Board eligible. Group or partnership—individual or multispecialty. Available July 1986.

Hal Tobias, M.D., 19456 NE 26th Ave., Apt. 14, North Miami Beach, FL 33180. Sackler (Israel) 1981. Board eligible. Group, partnership, solo. Available.

OBSTETRICS/GYNECOLOGY—Alan Cohen, M.D., 2335 Charney Rd., University Heights, OH 44118. Dominica 1982. Board eligible. Group, partnership, solo. Available July 1986.

D. L. Johnson, M.D., 7216 Montrose Rd., Woodbury, MN 55125. Minnesota 1977. Board eligible. Group or partnership. Available.

OPHTHALMOLOGY—Kenneth R. Brein, M.D., 55 Beacon St., Pittsburgh, PA 15217. University of Pennsylvania 1982. Board eligible. Group, partnership, solo. Available July 1986.

Andrew R. Gewirtz, M.D., 64 Pond Rd., Apt. G3, Woodbury, NY 11797. Chicago 1978. Board eligible. Board certified (PED). Single or multispecialty group. Available.

PULMONARY—Raymond Mantovani, M.D., 89 Fleet Pl., Mineola, NY 11501. Universidad Centro de Estudios Teenicos (Dominican Republic) 1981. Board eligible. Group, partnership, solo, hospital based. Available July 1986.

Fred A. Wagshul, M.D., 55 Mill Plain Rd., Unit 16-4, Danbury, CT 06811. Universidad Central del Este 1980. Board eligible (IM). Group, partnership, hospital based. Available July 1986.

RADIOLOGY—Jesus S. Santos, Jr., M.D., RD 2, Gibson Rd., Goshen, NY 10924. Santo Tomas (Philippines) 1960. Board certified. Available.

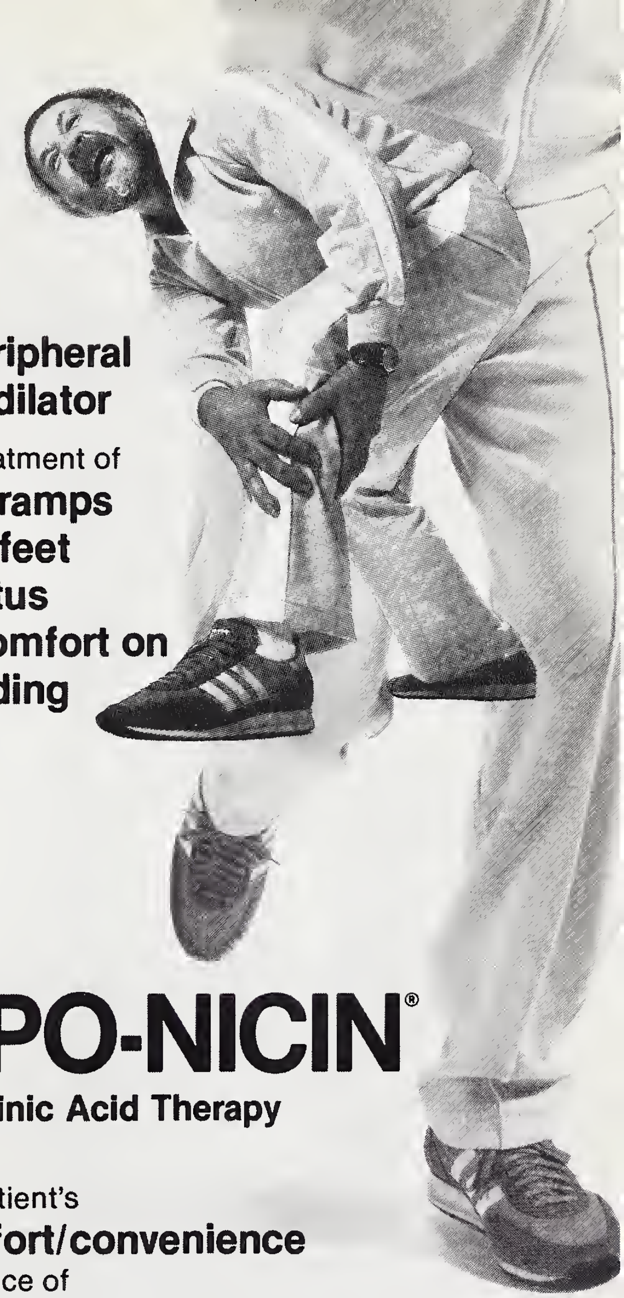
SURGERY, GENERAL—Bernard Henson, M.D., 1275 15th St., Apt. 16-0, Fort Lee, NJ 07024. Guadalajara (Mexico) 1979. Board eligible. Partnership or group. Available July 1986.

Derrick John Martin, M.D., 13-2 Granada Cres., White Plains, NY 10603. West Indies 1977. Board certified. Group, partnership, solo. Available.

Russell H. Zelman, M.D., 309 DeMott St., Mineola, NY 11501. Universidad del Noreste (Mexico) 1980. Board eligible. Group, solo, partnership, Available July 1986.

SURGERY, PLASTIC—Jerrold Zeitel, M.D., 240 E. Montgomery Ave., Apt. 16, Ardmore, PA 19003. Pennsylvania 1980. Board eligible. Group or partnership. Available July 1987.

UROLOGY—Louis C. Galdieri, M.D., 1563 Hampton Knoll Dr., Akron, OH 44313. Guadalajara (Mexico) 1979. Group or partnership. Available August 1986.



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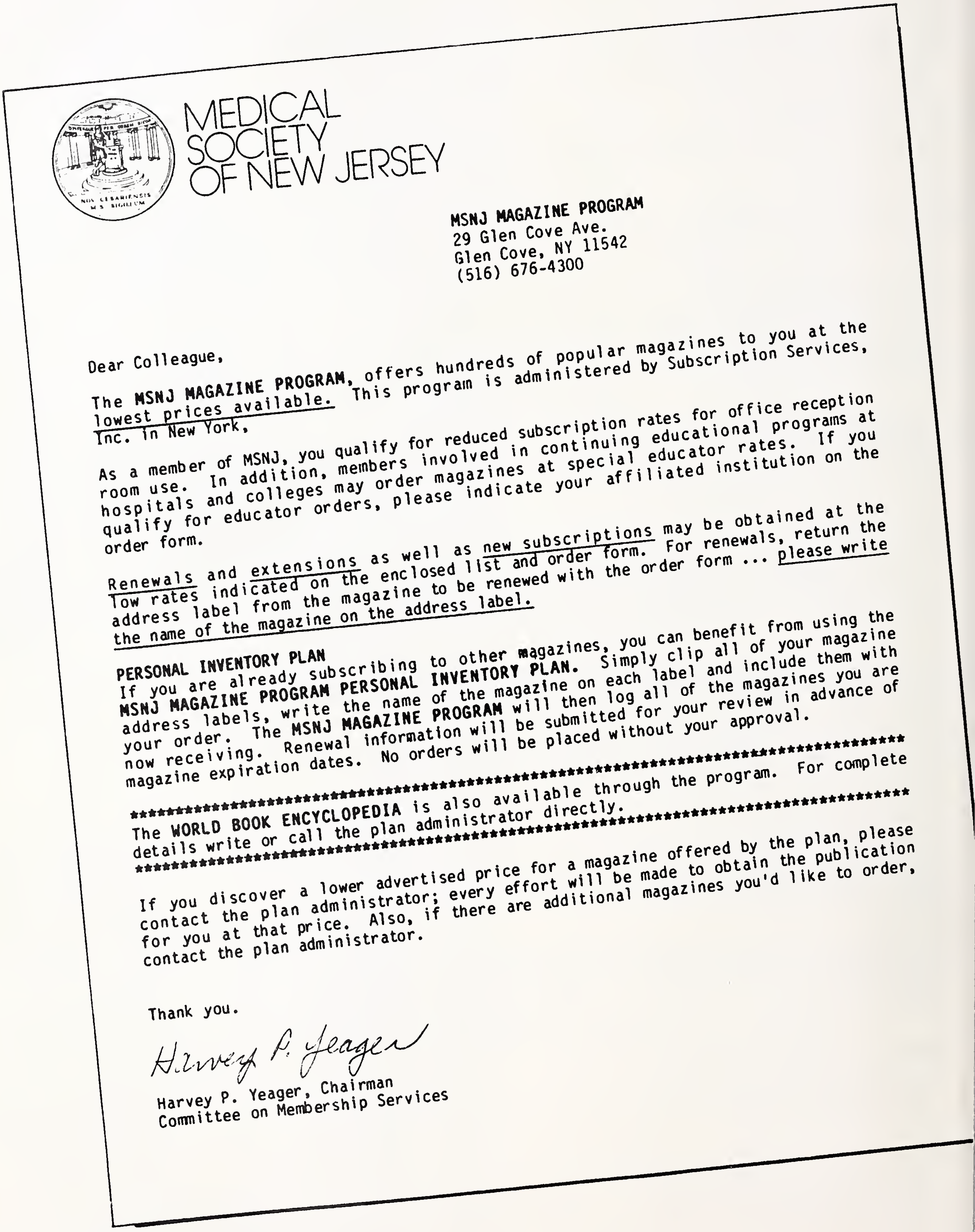

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American Heritage(6)	24.00	18.00	18.00	
Amer.Photographer(12)	19.90	9.95	6.65	8
Americana(6)	11.95	11.95	9.95	
ArtNews(12)	29.95	25.95	25.95	
Artist's Mag(12)	21.00	21.00	9.97	9
Atlantic Monthly(12)	9.95	9.95	9.95	
Audio(12)	17.94	8.97	8.97	
Baseball Digest(12)	14.95	14.95	7.97	10
Basketball Digest(8)	9.95	9.95	7.97	
#Better Hms-Gdns(12)	12.97	6.49	12.97	
#Bicycling(10)	15.97	8.00	12.97	
Black Enterprise(12)	15.00	9.00	7.50	
Bon Appetit(12)	15.00	15.00	15.00	
Boys Life(12)	13.20	13.20	11.97	
Car & Driver(12)	17.00	11.99	11.99	
Cats (12)	17.50	17.50	12.50	
Cat Fancy(12)	17.97	17.97	12.97	
Changing Times(12)	15.00	9.97	9.97	
Child Life:age 6-10(8)	11.95	11.95	9.97	
Childrens Dig:7-11(8)	11.95	11.95	9.97	
Commentary(12)	33.00	33.00	33.00	
#Connoisseur(12)	19.95	10.00	19.95	
Consumers Digest	14.97	14.97	10.97	
Consumers Reports(12)	16.00	16.00	16.00	
#COSMOPOLITAN(12)	**24.97**	**15.00**	**24.97**	
Cruising World(12)	18.00	18.00	14.00	
#Discover(12)	24.00	13.00	14.95	
Dog Fancy(12)	17.97	17.97	12.97	
Downbeat(12)	15.75	15.75	7.95	
Ebony(12)	16.00	14.00	9.97	
The Economist(52)	85.00	51.00	51.00	
Ellery Queen Myst(13)	22.75	19.50	9.97	10
Esquire(12)	17.94	9.95	9.95	
Essence(12)	12.00	9.96	9.00	
Family Circle(17)	14.97	14.97		
Family Computing(12)	19.97	11.97	11.97	
Family Handyman(10)	9.95	9.95	5.95	
Field & Stream(12)	13.94	7.94	7.94	
Fifty Plus(12)	15.00	11.97	11.97	
Financial World(26)	41.95	24.94	18.95	18
Food and Wine(12)	18.00	9.00	15.00	
Football Digest(12)	12.95	12.95	7.97	10
Forbes(28)	42.00	42.00	28.00	
Fortune(26)	42.00	21.00	21.00	
Games Magazine(12)	15.97	15.97	15.97	
Gentlemens Quart.(12)	18.00	13.50		
Glamour(12)	15.00	12.00		
Golf Digest(12)	19.94	11.97	11.97	
Golf Magazine(12)	15.94	8.97	7.97	

PUBLICATION (No. of iss. per yr)	REG. SUBSCR. PRICE	YOUR OFFICE PRICE	YOUR EDUC. PRICE	(No of iss)
#Good Housekeeping(12)	14.97	9.97	14.97	
Gourmet(12)	18.00	13.50	15.00	
#Harpers Bazaar(12)	16.97	8.97	16.97	
Harpers Magazine(12)	18.00	9.97	9.97	
Health(12)	22.00	13.32	11.00	
High Fidelity(12)	13.95	6.98	6.98	
Home(12)	15.00	15.00	15.00	
Home Mechanics (12)	11.94	8.97	8.97	
The Homeowner(10)	18.00	13.97	9.00	
Hot Rod(12)	13.94	8.97	8.97	
House and Garden(12)	24.00	18.00		
#House Beautiful(12)	15.97	7.99	15.97	
Humpty Dumpty:4-7(8)	11.95	9.97	9.97	
Jerusalem Post(24)	18.46	18.46	15.97	
Jet(52)	36.00	30.00	26.00	
Ladies Home Jrnl(12)	20.00	15.00	10.00	
#Life(12)	32.50	16.25	25.00	10
M:Civilized Man(12)	24.00	19.95	17.95	10
Mademoiselle(12)	15.00	12.00		
#Mccalls(12)	11.95	11.95	11.95	
Metropolitan Home(12)	15.00	7.50	7.50	
Modern Photography(12)	13.98	7.98	6.99	
#Money(12)	29.95	15.00	19.95	
Mother Jones(12)	18.00	12.00	12.00	
Motor Trend(12)	13.94	7.97	7.97	
MS Magazine(12)	16.00	10.97	10.97	
The Nation (52)	28.00	28.00	9.95	24
Nation's Business(12)	22.00	12.97	12.97	
Natural History(12)	20.00	15.95	15.95	
N.E.Journal of Medicine(52)		60.00		
New Republic (52)	48.00	48.00	28.00	
#New Shelter(9)	10.97	5.50	9.97	
N.Y. Rev.of Books(17)	28.00	28.00	25.95	
New York Mag.(52)	33.00	29.00	19.98	
New Yorker(52)	32.00	32.00	20.00	
#Newsweek(52)	41.00	21.80	21.80	
Omni(12)	24.00	15.96	15.96	
1001 Home Ideas(12)	22.00	15.97	11.00	
Organic Gardening(12)	12.97	9.97	9.97	
Outdoor Life(12)	13.94	7.97	7.97	
Outside(10)	16.00	12.00	8.97	
Ovation(12)	16.00	8.00	8.00	
PARENTS(12)	18.00	9.00	11.95	
Penthouse(12)	36.00	30.00	30.00	
People(52)	56.50	56.50	28.25	
Personal Computing(12)	18.00	18.00	8.97	9
Petersens Photog(12)	13.94	6.97	6.97	
Playboy(12)	25.00	21.50	21.50	
#Popular Mechanics(12)	13.97	7.00	13.97	
Popular Photogr.(12)	11.97	5.99	5.99	

PUBLICATION (No. of iss. per yr)	REG. SUBSCR. PRICE	YOUR OFFICE PRICE	YOUR EDUC. PRICE	(No of iss)
Popular Science(12)	13.94	7.97	7.97	
#Prevention(12)	13.97	7.00	13.97	
Psychology Today(12)	15.99	8.00	12.97	
Reader's Digest(12)	15.41	15.41	15.41	
#Redbook(12)	11.97	6.97	11.97	
Road and Track(12)	17.94	11.99	12.99	
Rolling Stone(26)	19.95	19.95	17.95	
The Runner(12)	16.97	12.97	8.97	
#Runner's World(12)	19.95	10.00	12.97	
Sail(12)	21.75	21.75	21.75	
Saturday Eve.Post(9)	12.97	12.97	12.97	
Savvy(12)	12.00	12.00	6.00	
Science '86(11)	18.00	18.00	13.95	
#Science Digest(12)	13.97	6.99	13.97	
Scientific Amer.(12)	24.00	24.00	24.00	
Self(12)	15.00	12.00	N.A.	
Seventeen(12)	13.95	13.95	13.95	
Shape(12)	20.00	20.00	8.97	6
Ski(8)	11.94	6.97	6.97	
Skiing(7)	9.98	4.99	4.99	7
Skin Diver(12)	13.94	7.97	7.97	
Sport(12)	12.00	7.97	7.97	
Sporting News(24:6 mo)	22.50	11.97	11.97	
#Sports Afield(12)	13.97	6.99	13.97	
#Sports Illustr.(54)	56.50	34.00	29.45	
Stereo Review(12)	9.97	4.99	4.99	
Teen(12)	12.95	7.95	7.95	
Tennis(12)	17.94	8.97	8.97	
#Time(52)	58.25	29.25	28.00	50
#Town & Country(12)	24.00	12.00	24.00	
Travel & Leisure(12)	25.00	12.50	25.00	
TV Guide(52)	31.20	31.20	26.00	
#U.S.News/Wrld.Rep(52)	41.00	20.50	20.50	
Vegetarian Times(12)	19.95	14.95	14.95	
Video(13)	15.00	15.00	7.50	
Video Review(12)	12.00	6.97	6.97	
Village Voice(52)	32.76	32.76	22.00	
Vogue(12)	24.00	21.00		
W Magazine(26)	26.00	23.00	17.95	
Weight Watchers(12)	13.97	11.97	11.97	
Woman's Day(17)	15.13	15.13	15.13	
Women's Sports(12)	12.95	12.95	8.95	
Workbench(6)	6.00	6.00	5.00	
Working Mother(12)	11.95	9.95	9.95	
Working Woman(12)	18.00	12.00	15.00	
World Press Review(12)	16.95	16.95	14.98	
Writer's Dig.(9 iss)	18.00	9.97	9.97	
Yankee (Colonial)(12)	15.00	15.00	12.97	
YM(12)	14.00	10.95	10.95	

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NJMD186

New Jersey Society of Pathologists/ Pennsylvania Association of Clinical Pathologists

Joint Annual Meeting

THURSDAY-SATURDAY
May 15-17 1986

HYATT CHERRY HILL, Cherry Hill, NJ

For further information please contact:
Cathy Gillmer
Executive Secretary
(609) 896-1717

ACUPUNCTURE IN CLINICAL PRACTICE

N.Y. State Boards of Medicine & Dentistry 25-hour accredited seminar and workshop on the latest theories & techniques of manual & electroacupuncture and TENS, applicable toward the 200-hour requirement for certification, will be given for licensed clinicians (with or without prior training) during weekend, May 30-31-June 1, Mulford Plaza Hotel, New York City, NY. Co-sponsored by the International College of Acupuncture & Electro-Therapeutics, its official journal, Acupuncture & Electro-Therap. Res., Int.J. (published by Pergamon Press and Indexed in 15 major indexing periodicals; INDEX MEDICUS, etc.), the Heart Disease Research Foundation and the Neuroscience Dept. of Long Island College Hospital, Pharmacology Dept. of The Chicago Medical School. Also eligible for AMA/CME credit. For information, contact Y. Omura, M.D., ScD., 800 Riverside Drive (8-1), NYC 10032. Tel: (212) 781-6262 or (212) WA8-0658, Phil Shinick, Ph.D., Tel: (201) 246-8557. SECOND INTERNATIONAL SYMPOSIUM—OCT. 15-19, 1986.

RANDY L. FRIDKIS AUCTIONS INC.
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RANDY L. FRIDKIS AUCTIONS INC., AS AGENT

TUESDAY MAY 20, 1986 AT 11AM

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Academy of Medicine of New Jersey

invites you to attend its

ANNUAL AWARDS DINNER

WEDNESDAY, MAY 28, 1986
6:00 P.M.

CHANTICLER
500 Millburn Avenue, Short Hills, New Jersey

AWARD RECIPIENTS

Edward J. Ill Award
to
PALMA E. FORMICA, M.D.

Citizens Award
to
MALCOLM A. BORG

For further information on dinner and table reservations contact the Executive Offices of the Academy of Medicine of New Jersey

Two Princess Road
Lawrenceville, New Jersey 08648
609-896-1717

CME CALENDAR

The following is a list of continuing medical education courses for the next two months. Contact the sponsoring organization for further information.

This list is compiled through the cooperation of the Committee on Medical Education of the Medical Society of New Jersey, The Academy of Medicine of New Jersey, the New Jersey Chapter of the American Academy of Family Physicians, and the Office of Continuing Medical Education of the UMDNJ. For information on accreditation, please contact the sponsoring organization(s), indicated by italics—last line of each item.

ANESTHESIOLOGY

June

2 **Anesthesia Lectures**
9 7:45 A.M.—Auditorium, 3rd Fl.,
16 Newark Beth Israel Medical Center
23 *(UMDNJ)*
30

2 **Anesthesia Lectures**
9 7 A.M.—Medical Science Bldg.,
16 E-545, NJ Medical School, Newark
23 *(UMDNJ)*
30

3 **Anesthesia Lectures**
10 4 P.M.—Medical Science Bldg.,
17 E-545, NJ Medical School, Newark
24 *(UMDNJ)*

25 **Anesthesia Grand Rounds**
4-5 P.M.—University Hospital,
H-350, Newark
(UMDNJ)

CARDIOLOGY

June

27 **Cardiothoracic Journal Club**
7:30 P.M.—13 Ridgeway Drive,
West Orange
(UMDNJ)

MEDICINE

June

Mini-Residency in Proctology and Disease of the Colon
One week commitment—Contact
Overlook Hospital, Summit
(Overlook Hospital and AMNJ)

2 **Blood Banking Conference**
9 11 A.M.—Medical Science Bldg.,
16 C-128, NJ Medical School, Newark
23 *(UMDNJ)*
24
30

3 **Ototoxic Drugs**
7-8 P.M.—West Hudson Hospital,
Kearny
(West Hudson Hospital and AMNJ)

3 **Pulmonary Conference**
10 8-9 A.M.—University Hospital,
17 Newark
24 *(UMDNJ)*

4 **Medical Lecture Series**
11 10:30 A.M.-12 noon—Christ
Hospital, Jersey City
(Christ Hospital and AMNJ)

4 **Rehabilitation Medicine**
11 4:15-5:15 P.M.—Kessler Institute,
18 West Orange
25 *(UMDNJ)*

4 **CDS Prescribing Practices**
10:30 A.M.-12 noon—St. Mary's
Hospital, Passaic
(St. Mary's Hospital and AMNJ)

4 **Update on Anal and Colorectal Diseases**
1-5 P.M.—The Manor, West Orange
(NJ Gastroenterological Society and AMNJ)

5 **Trauma Conference**
12 7:45-8:45 A.M.—University Hospital,
19 H-349, Newark
26 *(UMDNJ)*

5 **Digestive Disease Case Report**
12 8-9 A.M.—Medical Science Bldg.,
19 H-249, NJ Medical School, Newark
26 *(UMDNJ)*

5 **Digestive Disease Research and**
12 **Literature Review Conference**
12 9-10 A.M.—Medical Science Bldg.,
26 H-249, NJ Medical School, Newark
(UMDNJ)

5 **Allergy/Immunology Lecture**
12 11 A.M.-12 noon—United Hospital,
19 Newark
26 *(UMDNJ)*

6 **Cancer Research Colloquium**
13 12 noon-1 P.M.—Medical Science
20 Bldg., G-506, NJ Medical School,
27 Newark
(UMDNJ)

6 **Dept. of Medicine Grand Rounds**
13 12 noon-1 P.M.—Medical Science
20 Bldg., B-610, NJ Medical School,
27 Newark
(UMDNJ)

6 **Urology Basic Surgery Conference**
13 2-3 P.M.—Medical Science Bldg.,
20 C-600, NJ Medical School, Newark
27 *(UMDNJ)*

6 **Urology Grand Rounds**
13 3-5 P.M.—Medical Science Bldg.,
20 C-600, NJ Medical School, Newark
27 *(UMDNJ)*

6 **Renal Conferences in Nephrology**
20 4-5 P.M.—University Hospital,
H-349, Newark
(UMDNJ and AMNJ)

10 **An Overview of Cancer Pain**
17 **Drug-Induced Hepatic Toxicity**
9-10 A.M.—Holy Name Hospital,
Teaneck
(Holy Name Hospital and AMNJ)

13 **Current Clinical Concepts in Rheumatology/Immunology**
12:15-1:15 P.M.—Kennedy Memorial
Hospital, Stratford
(UMDNJ)

16 **IVP Conference**
5-9 P.M.—East Orange VA Hospital,
Rm. 142
(UMDNJ)

18 **Interhospital Rheumatology Rounds**
4:30-6:30 P.M.—University Hospital,
I-355, Newark
(UMDNJ)

19 **Managing Hepatitis**
5-6:30 P.M.—Somerset Medical
Center, Somerville
(Somerset Medical Center and AMNJ)

19 **Managing Hepatitis**
5-7:30 P.M.—Somerset Medical
Center, Somerville
(Somerset Medical Center and AMNJ)

NEUROLOGY/PSYCHIATRY

June

2 **Pervasive Developmental Disorder Grown Up**
8:15-10:15 P.M.—1046 South
Orange Ave., Short Hills
(Essex Psychiatric Seminar and AMNJ)

4 **Grand Rounds in Psychiatry**
10:30 A.M.-12:30 P.M.—Medical
Science Bldg., 610, NJ Medical
School, Newark
(UMDNJ)

5 **Case Seminars**
19 8-10 P.M.—312 Harding Drive,
South Orange
(Advanced Psychiatric Study Group and AMNJ)

7 **The Adolescent Family: Teenagers and Their Children**
9 A.M.-4 P.M.—NJ Medical School,
Newark
(UMDNJ and AMNJ)

18 **Psychiatric Lecture**
10:30 A.M.-12:30 P.M.—Medical
Science Bldg., B-610, NJ Medical
School, Newark
(UMDNJ)

18 **Psychiatry Lecture**
1-3 P.M.—Essex County Hospital
Center, Cedar Grove
(UMDNJ)

PATHOLOGY

June

3 **Anatomic Pathology**
10 5:15 P.M.—Medical Science Bldg.,
17 C-555, NJ Medical School, Newark
24 *(UMDNJ)*

Polk County Medical Association
SPRING SEMINAR

Grenelefe Resort — (Haines City) Florida

Convenient to both the Orlando (closer) and Tampa (approximately one hour) airports. Grenelefe is centered in Florida's "Vacation Wonderland." Within 30-45 minutes driving time to Walt Disney World's Epcot Center and Magic Kingdom, Cypress Gardens, Sea World and Circus World. If you're driving take I-4 to U.S. 27, south 12 miles to SR 546 East.

Friday, Saturday and Sunday
May 16, 17 and 18, 1986

This seminar has been designed for physicians, office staff, hospital nursing and administrative staffs as well as technicians, medical students, interns and residents. There is no registration fee for allied health personnel but registration is imperative.

Continuing Medical Eduation: AMA Category I — 11 hours; this program has been reviewed and is acceptable for 10½ Prescribed hours by the American Academy of Family Physicians; nursing credit applied for.

— PROGRAM —

Friday, May 16

Stanley C. White, MD, Senior Scientist, Biomedical and Environmental Laboratories, Kennedy Space Center, Satellite Beach, FL

Saturday, May 17

Disciplining the Impaired Physician

Joseph Lawrence, Esquire, Chief Attorney and Director, Division of Regulation, Department of Professional Regulation, Tallahassee, FL

J. Darrell Shea, MD, Chairman, Florida Board of Medical Examiners, Orlando, FL

Donald Weidner, Esquire, Associate General Counsel, Florida Medical Association, Jacksonville, FL

Roger A. Goetz, MD, Medical Director, Florida Medical Foundation Impaired Physicians Program, Jacksonville, FL

Risk Management

Patrice Taverniere, Senior Health Care Risk Management Representative, St. Paul Insurance; Chairman, Health Care Risk Management Advisory Committee for Department of Insurance, State of Florida, Orlando, FL

Leilani Kicklighter, Risk Management, Miami General Hospital, President, Florida Society for Hospital Risk Management, Miami, FL

Robert C. McCurdy, Esquire, Staff Attorney and House Counsel, Lee Memorial Hospital, Fort Myers, FL

John C. Kruse, MD, Anesthesiology, Jacksonville, FL

Sunday, May 18

James Sammons, MD, AMA Executive Vice-President

Neurology of Golden Years

Melvin Greer, MD, Chairman, Department of Neurology, University of Florida, Gainesville, FL

Peritz Scheinberg, MD, Chairman, Department of Neurology, University of Miami, FL

Daniel Traviesa, MD, Neurologist, Watson Clinic, Lakeland, FL

Co-sponsored by the Florida Hospital Association, Lakeland Regional Medical Center, Winter Haven Hospital, Lake Wales Hospital, Bartow Memorial Hospital and Heart of Florida Hospital.

REGISTRATION FEE

Physicians: $185.00

Allied Health: $85.00
(no registration fee — meals only)

Please make check payable to PCMA and mail to: **Polk County Medical Association, P.O. Box 927, Lakeland, FL 33802, (813) 682-0543.**

POLK COUNTY MEDICAL ASSOCIATION SPRING SEMINAR
May 16 - 18, 1986
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- *If room requested is not available, reservation will be made at the nearest available rate.*
- *Special room requests will be honored whenever possible.*
- *The above rates will be honored two days prior and two days after your meeting upon request — subject to availability.*
- *Room rates are subject to 5% state tax.*

In order to guarantee your reservation, please enclose a one-night's deposit, including tax. Reservations received without a deposit will not be confirmed.

Name(s) ____________________

Address ____________________

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Home Phone ____________________ Arrival ____________ Check In Time is 3:00 p.m.

Business Phone ____________________ Departure ____________ Check Out Time is 11:00 a.m.

Arrival By ☐ Car ☐ Air *(Deposit Refundable If Cancellation Received 7 Days Prior to Arrival Date)*

4 **Neuropathology Conference**
18 10:30 A.M.-12:30 P.M.—Medical
Science Bldg., C-555, NJ Medical
School, Newark
(UMDNJ)

4 **Pathology Experimental Seminar**
18 12 noon-1 P.M.—Medical Science
Bldg., C-555, NJ Medical School,
Newark
(UMDNJ)

4 **Pathology Grand Rounds**
11 1-5 P.M.—Medical Science Bldg.,
18 C-555, NJ Medical School, Newark
25 *(UMDNJ)*

5 **Pathology/Radiology/Orthopedics**
12 **Conferences**
19 8-9 A.M.—University Hospital,
26 Newark
(UMDNJ)

5 **Pathology Experimental Seminar**
12 12 noon-1 P.M.—Medical Science
19 Bldg., C-555, NJ Medical School,
26 Newark
(UMDNJ)

28 **Soft Tissue Workshop**
29 8 A.M.-4:30 P.M.—NJ Medical School,
Newark
(UMDNJ and AMNJ)

PEDIATRICS

June

6 **Advances in Pediatrics**
13 9:30-10:30 A.M.—Medical Science
20 Bldg., B-610, NJ Medical School,
27 Newark
(UMDNJ)

RADIOLOGY

June

4 **Radiology Grand Rounds**
18 2-4 P.M.—University Hospital,
H-346, Newark
(UMDNJ)

5 **Pathology/Radiology/Orthopedics**
12 **Conferences**
19 8-9 A.M.—University Hospital,
26 Newark
(UMDNJ)

SURGERY

June

2 **Tumor Conference**
9 8-9 A.M.—University Hospital,
16 H-349, Newark
23 *(UMDNJ)*
30

7 **Surgical Treatment of**
Cardiothoracic Diseases
10 A.M.-12 noon—Medical Science
Bldg., G-506, NJ Medical School,
Newark
(UMDNJ)

7 **Surgical Morbidity and Mortality**
14 **Conference**
21 8:30-9:30 A.M.—Medical Science
28 Bldg., G-506, NJ Medical School,
Newark
(UMDNJ)

19 **Interventional Endoscope**
5-6 P.M.—Shore Memorial Hospital,
Somers Point
(Shore Memorial Hospital and
AMNJ)

July

7 **Surgical Morbidity and Mortality**
14 **Conference**
21 4:30-6:30 P.M.—Medical Science
28 Bldg., B-610, NJ Medical School
(UMDNJ)

SURGICAL SPECIALTIES

June

2 **Orthopedic Grand Rounds**
9 6-8 P.M.—Medical Science Bldg.,
16 B-556, NJ Medical School, Newark
23 *(UMDNJ)*
30

5 **Pathology/Radiology/Orthopedics**
12 **Conferences**
19 8-9 A.M.—University Hospital,
26 Newark
(UMDNJ)

12 **Neurosurgery Lecture**
4-6 P.M.—University Hospital,
E-343, Newark
(UMDNJ)

18 **Grand Rounds in Ophthalmology**
7-9 P.M.—United Hospitals Annex
Bldg., Newark
(UMDNJ)

MISCELLANEOUS

June

11 **Office Management of Common**
Medical Problems
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LETTERS TO THE EDITOR

Conroy Discussions

Dear Dr. Krosnick:

In the December 1985 issue of *NEW JERSEY MEDICINE* devoted to the Conroy decision, two papers were presented under the classification of "Opinion." If an opinion is based on misinterpretation of the facts, then the reader easily could be misled into arriving at erroneous conclusions. A closer examination of these two papers leads me to the conclusion that the writers did not read the printed opinion of the New Jersey Supreme Court. If they did read it, they have completely distorted its intent and meaning.

Professor McIntyre states the following: "The Conroy decision, however, assigns no role whatsoever to such an institutional ethics committee. What it does instead is to create a geriatric version of the Baby Doe squads by assigning an investigatory role to the State Office of the Ombudsman whenever a physician or family wants to withhold or withdraw life-sustaining medical therapy from an elderly nursing home patient who is certified to be terminally ill."

The Supreme Court itself stated: "The Court's holding is restricted to nursing home residents like Claire Conroy who are suffering from serious and permanent mental and physical impairments, who will probably die within approximately one year even with treatment, and who, though formerly competent, are now incompetent to make decisions about their life-sustaining treatment and are unlikely to regain such competence."

In another situation, Doctor McIntyre declares: "Absent specific legislation, would a decision by a patient reached about terminating treatment while legally competent, i.e. a living will, be legally binding when the patient becomes incompetent?"

The answer to his question is found in the Court decision. The Court clearly noted that a clear expression by the patient, either in a "living will" or some other writing or verbally would be legally binding. It also is noted that the patient might have executed a power of attorney or a proxy authorizing someone else to make the decisions for him—if he cannot make them himself.

How Doctor McIntyre could have read the decision and missed that lengthy discourse is puzzling.

Doctor McIntyre also wrote: "Since the Conroy decision was handed down in January 1985, several key individuals in New Jersey have called upon Governor Kean and the legislature to establish a blue ribbon panel to evaluate the needs of dying patients and their families and to make recommendations on a series of legislative initiatives which would protect the rights of all persons while preserving the state's interest in protecting and preserving life."

THE ACTUAL CHRONOLOGY

January 17, 1985. Conroy matter decided.

February 8, 1985. Panel sponsored by Citizens Committee on Biomedical Ethics meeting in Princeton. A Federal judge (then the Public Advocate) calls for enabling legislation for governor's Commission to study legal and ethical problems in delivery of health care.

February 22, 1985. Assembly Bill No. 3316—An act creating a Commission to study the legal and ethical problems in the delivery of health care introduced in the Assembly.

November 12, 1985. Assembly Bill No. 3316 signed into law by Governor Kean.

COMMENTARY

Doctor Nevins also seems to have missed several points. "It is necessary and desirable to involve the Office of the Ombudsman, as suggested by the Supreme Court, into the decision-making process when artificial life support (included feeding) would be terminated or not initiated in nursing home patients."

This was not the issue in Conroy as decided by the State Supreme Court. The Court's language is far more precise and limited than that of Doctor Nevins. The Supreme Court decision stated, "Medical evidence bearing on the patient's condition, treatment, and prognosis, like evidence of the patient's wishes, is an essential prerequisite to decision making under the subjective test. The medical evidence must establish that the patient fits within the Claire Conroy pattern: an elderly, incompetent nursing home resident with severe and permanent mental and physical impairments and a life expectancy of approximately one year or less."

Dr. Nevins adds the following comments: "Analysis of the replies to the statement about the Ombudsman's role reveals that the only physician who strongly favored the Court's recommendation was an academic cardiologist who has no contact with nursing home patients. Conversely, 33 respondents indicated that they often cared for this category of patients and this subgroup unanimously opposed the Court's plan; 5 disagreed, 29 disagree strongly."

I strongly urge that this journal apologize for printing such an insulting statement and that Doctor Nevins apologize for its writing.

Finally, we should hear from the New Jersey Association of Health Care Facilities. *The Proposed Guidelines for Supportive Care*, approved by MSNJ, were forwarded to the foregoing Association which represents the nursing homes of New Jersey. The guidelines recommended an intramural equivalent of an Ethics Committee.

Upon review of these *Guidelines*, the Association's Right to Live/Die Task Force accepted the *Guidelines* as, and if, amended. "Our Association's Right to Live/Die Task Force has reviewed these *Guidelines* and has recommended some amendments. Our amended version of your document is enclosed for your review. The New Jersey Association of Health Care Facilities has voted endorsement of this amended version. If the Medical Society agrees with our amended version, that endorsement will continue. Otherwise the matter would have to be resubmitted to us for further consideration."

Guidelines for Supportive Care Plans for Nursing Home Residents as amended by NJAHCF Task Force state, "8. [Facilities shall establish a Supportive Care Plan Review Committee to examine and evaluate every SCP decision.] *The Ombudsman for the Institutionalized Elderly will examine and evaluate every SCP decision, as per Supreme Court of New Jersey Opinion A-108, Conroy; and 9. The basic goals shall be full discussion, full understanding, full agreement, full documentation, and full review. [.] by the Ombudsman for the Institutionalized Elderly.*" (Matter enclosed in brackets [thus] in the foregoing is intended to be omitted. Matter italicized is amended material.)

This letter represents my personal response to the opinions presented in the December 1985 issue of *NEW JERSEY MEDICINE.* The materials presented are from the Society files and, of course, the Supreme Court decision. This letter in no way represents the thinking of the Committee on Biomedical Ethics or the Board of Trustees of the Medical Society of New Jersey.

It is important that the reader of an opinion be assured that the opinion he is reading is based upon facts.

(signed) David Eckstein, M.D.

RESPONSES

March 2, 1986

Dear Dr. Krosnick,

Thank you for the opportunity to review and respond to the criticisms by Dr. David Eckstein to my article on the Conroy decision that appeared in the December 1985 issue of *NEW JERSEY MEDICINE.* I have identified three criticisms of my article in his letter.

The first criticism involves what I believe to be the scope of the Conroy decision and the role of the ombudsman. The decision applies only to "nursing home residents like Claire Conroy," but what Dr. Eckstein fails to realize is that it is only the ombudsman who can decide whether or not the patient fits the description of the Conroy situation. I read this to mean that the ombudsman is to be called in whenever: a) an incompetent, elderly (age 62 or over) nursing home patient is "suffering from serious and permanent mental and physical impairments"; b) it is felt that the patient would "probably die within approximately one year even with treatment"; c) the physician or family believes that life-prolonging medical treatment (including nutrition and hydration) no longer is in the patient's best interest. The mechanism created by the Conroy decision then requires a guardianship proceeding or court review if guardianship already has been assigned.

Dr. Eckstein's second criticism simply misses the point of my argument. He has picked a rhetorical question out of my introduction without looking for the answer in the section in which I criticize the "subjective test." The Conroy Court talked very favorably about a patient's right to make an advanced directive (living will), but it also set up stringent criteria for judging whether that "living will" would be considered valid at the time it would be acted upon. Page 947 has the answer Dr. Eckstein missed: "The Court ... required that all of the ... criteria for the subjective test (including the living will) be judged by qualifying standards of remoteness, consistency, thoughtfulness, the maturity of the patient at the time the declaration was made, and specificity. Two sentences later, I forecast a possible consequence and state that "even a signed living will could be disqualified if it failed the Court's measure for being not recent enough, not logically consistent, or not specific enough; to say nothing about how one would judge the patient's thoughtfulness or level of maturity when the document was issued." One also must note here that it is up to the ombudsman to decide whether or not an incompetent patient's previously enacted "living will" was valid and determinative for decision making regarding life-prolonging medical therapy.

Dr. Eckstein's final comment on my article pertains to the chronology of events since the Conroy decision was handed down in January 1985. He insinuates that I simply have not kept up with current events. But, in fact, I was the first speaker on the Citizen's Committee program at the Woodrow Wilson School at Princeton University in February 1985 and was the first to call for the establishment of the state-wide commission for the study of ethical and legal problems in the delivery of health care. What Dr. Eckstein does not realize is that my Conroy article was written and submitted to *NEW JERSEY MEDICINE* in March 1985 and was published in December 1985.

Since the Commission is to become a reality in 1986, it is my hope that they will look into all of the facts surrounding the Conroy decision and examine the appropriateness and workability of the specific guidelines, especially the role of the ombudsman in light of the case of Hilda Peter, the single case that has followed the Conroy guidelines. I regard this investigatory role for the ombudsman as an unwarranted and indelicate intrusion into the physician-patient-family-nursing home relationship, but would be anxious to read the report of the new Commission.

(signed) Russell L. McIntyre, Th.D.

March 3, 1986

Dear Dr. Krosnick:

I fear that it will be difficult for the casual reader to evaluate objectively the merits of Dr. Eckstein's critique of my and Dr. McIntyre's articles unless they have themselves read the more than 100-page Conroy decision. Nonetheless, the issues are of profound importance since they address the fundamental role of the physician as clinical decision maker.

It intrigues me that Dr. Eckstein's criticisms serve only to substantiate my own thesis. He takes me to task for interpreting the Conroy decision too broadly rather than as being applicable only in the narrow instance of feeding-tube decisions that closely approximate the Claire Conroy case. Yet, the response of the NJAHCF Task Force to his own Biomedical Ethics Committee's *Guidelines for Supportive Care* illustrates that the genie already is out of the bottle since they rejected outright the idea of supportive care plan committees in nursing homes in favor of ombudsman review a la Conroy. The reader should be aware that the supportive care plan proposal did not pertain to feeding tube decisions, but was intended to be applied generally to nursing home patients with terminal diseases. Although at the time of this writing more than one year after the Conroy decision, the ombudsman has yet to make a public ruling in any case, several informal inquiries by physicians to his office have indicated that he interprets his authority to include any instance of withholding medical treatment as a possible "abuse" with no distinction made between negligence and calculated reduction of intensity of care in dying patients. Lacking clarification to the contrary, presumably this would include failure to initiate aggressive treatment or hospitalizing an end-stage patient. The problem really is not how the ombudsman will decide in a given case, but rather, that the Court has given him the authority to be in the position of being the final arbiter.

Dr. Eckstein also objected to including the results of a survey that was designed to gauge the attitudes of internists about the Conroy decision. My intent, of course, was to demonstrate that those who work closest with nursing home patients appreciate the unsuitability and intimidating effect of prospective government review. The sophisticated reader will appreciate that all questionnaires inevitably are flawed, but I do believe that this survey validly represented the mainstream of thinking of New Jersey internists on this issue.

What is of greater concern to me is what is Dr. Eckstein's own opinion, not clearly stated in his letter. This is critical since he bears the heavy responsibility of representing Society members not only as chairman of its Committee on Biomedical Ethics, but now as one of only a very few physicians on the newly appointed 27-member Commission formed to advise the state legislature about future directions in health policy related to care of the dying. His voice should be unambiguous about the wisdom of government intervention at the bedside as well as about the growing tendency to undermine the physician's traditional role in favor of decision making by a multidisciplinary committee. Elements of this are present in the Weidel bill (A-776) now being heard in the Assembly and even in his own committee's *Guidelines for Supportive Care* that were approved by the Society in December 1984.

I hope that I do misunderstand the Conroy decision, but I fear that I do not. Certainly, it behooves all New Jersey physicians to educate themselves and to keep abreast of these issues which potentially could cause a Copernican reversal of roles about who best represents the patient's interests—government or physician?

(signed) Michael A. Nevins, M.D.

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Chandler and Grant's Glaucoma; Computed Tomography of the Head, Neck, and Spine; Critical Care Pediatrics; 1985 Year Book of Nuclear Medicine; Infection, Immunity, and Blood Transfusion Progress; Neuro-Ophthalmology

Chandler and Grant's Glaucoma, 3rd Edition

David L. Epstein, M.D. Philadelphia, PA, Lea & Febiger, 1986. Pp. 539. Illustrated.

Chandler and Grant's original book on glaucoma has been beautifully updated by Dr. Epstein. The easy readability of the original text has been maintained in a clear and detailed fashion, similar to the lecture style of Dr. Paul Chandler.

The subject matter of glaucoma is thoroughly and systematically approached and discussed. The book begins with a section within which the general considerations of glaucoma, examination of the eye in glaucoma, when to begin treatment, and laser methods are discussed.

The chapters also include the effect of environmental factors in glaucoma, as well as detailed discussions of virtually every aspect of the diagnosis and treatment of the various glaucomas in adults. The author includes the important current and controversial concept of ocular hypertension versus glaucoma and the importance of changes in the optic nerve as the earliest diagnostic sign as well as the place of visual fields, particularly the importance of suprathreshold automated perimeters.

A unique feature of this excellent book is the extensive review of the relationship of glaucoma to systemic diseases, especially vascular problems.

The author details the surgical procedures of glaucoma surgery and the treatment of complications thereof. The final chapter deals thoroughly with the diagnosis and treatment of glaucoma in childhood.

I heartily recommend this book for reference and as a primer on the subject of glaucoma.

Alfonse A. Cinotti, M.D.

Computed Tomography of the Head, Neck, and Spine

Richard E. Latchew (ed). Chicago, IL, Year Book Medical Publishers, 1985. Pp. 783. ($145)

This is a very good advanced neuroradiology review. It is divided into multiple sections dealing with physiology; cranial disease, including the sella; and diseases of the head and neck. The first section, devoted to physiology, includes a chapter on contrast material. The following chapters of this particular section, providing a review of the CT of the brain, especially are well written with pathological correlation. The scans are of good quality as well. A chapter entitled, "Differential Diagnosis of Intercranial Tumors," is a good attempt to evaluate intracranial masses.

While not quite as well written, the chapters on head and neck radiology also are informative. The chapter on the neck needs both additional rewriting and scans that are of an improved quality. The chapter on the spine is well organized with an abundance of good quality scans. Other chapters concerning dementia and cerebral blood flow fall more closely in the direction of research.

Generally, this book gives more information than the other basic texts, but its manner is as clear. It is recommended to the physician who wishes an indepth discussion of neuroradiology.

Neil B. Horner, M.D.

Critical Care Pediatrics

Sol S. Zimmerman, M.D., and Joan Holter, R.N. Philadelphia, PA, W.B. Saunders Company, 1985. Pp. 557. ($39.95)

This book was written to provide health-care givers information devoted to meeting the challenge of intensive care unit (ICU) pediatrics, exclusive of the newborn period.

The text differs from other emergency care publications in several respects. First, fairly comprehensive pathophysiology is included before therapies are addressed. Second, some illustrative case reports are included at the end of each chapter to enhance the text. Third, a suggestive reading list is added to complement the material presented. Fourth, the book is presented in textbook style, as opposed to handbook style. And lastly, the book is innovative in its physician/nurse dyad management approach. For this last reason, the authors are to be commended for their uniqueness. The nursing sections, included after those chapters dealing with specific ICU problems, are in outline form to evaluate the patient and parent needs or sensitivities. For those of us not attuned to nursing detail, it should be emphasized that the insight gained from this holistic approach is most rewarding.

Basically, the text is divided into four sections. The first section deals primarily with the "hi tech" aspects of critical care management. It includes topics basic to general principal care, e.g. fluids and electrolytes, shock, increased intracranial pressure. Section two deals with rather inclusive specific disease problems of the various systems. Section three deals primarily with burns and trauma. The last section deals with poisoning but, by necessity, is limited.

A book of this nature, although multiauthored, cannot be all-inclusive in scope. For example, topics such as sickle cell crisis, immune deficient states, and oncologic crises are omitted.

Critical Care Pediatrics is recommended reading for those physicians dealing with seriously ill children. For the primary care physicians not based in a tertiary care center, it provides invaluable information on available care. With this in mind, referral patterns to an "intensivist" should be established to enhance overall pediatric care.

Frank Vanore, M.D.

1985 Year Book of Nuclear Medicine

Paul B. Hoffer, John C. Gore, Alexander Gottschalk, Dirk Sostman, Barry Zaret (eds). Chicago, IL, Year

Book Medical Publishers, 1985. Pp. 424. ($44.95)

This book follows in the tradition of excellent synopses of the nuclear medicine literature. The 1985 editors include Drs. Gore and Sostman who were added for their expertise on magnetic resonance.

The editors have selected articles for each area of nuclear medicine such as cardiology, pulmonary, infectious disease, and physics. The endocrine system includes a nice review of thyroid therapy. The review of MRI (NMR) includes such topics as chemical shift and phosphate imaging. The text also includes the James L. Quinn essay—this year on radiolabeled antibodies.

I found this to be a well-done review with ample editorial comments. It is highly recommended to the physician who wishes to stay current in the field.

Neil B. Horner, M.D.

Infection, Immunity, and Blood Transfusion Progress in Clinical and Biological Research, Volume 182

Roger Y. Dodd and Lewellys F. Barker (eds). New York, NY, Allan R. Liss, Inc., 1985. Pp. 464. ($68)

This hardcover, reasonably priced book represents the proceedings of the XVI Annual Scientific Symposium of the American Red Cross, Washington, D.C., May 9 to 11, 1984, on the subject which is the title of the book. The symposium honored the late Wolf Szmuness, M.D. (1919 to 1982) who contributed to the development of hepatitis B vaccine.

There are 64 contributors and participants. The vast majority of the contributors were speakers at the symposium. In the preface it is noted that ten years have passed since the last Red Cross symposium devoted to transfusion-associated transmissable diseases. Since that time and with the awesome advances that have been made in biologic research, particularly in immunology, there is a vast amount of new information in this book.

There is a very informative preface, an introduction, two short speeches on the personality and work of Dr. Szmuness, and the nine sessions of the symposium: Transfusion Hepatitis: Unresolved Problems; Parasitic Diseases, Transfusion Associated; Herpesviruses; Human T-Cell Leukemia Virus (HTLV); New Diseases; New Challenges (AIDS); Aids and Transfusion; Immune Function and Blood Transfusion; and Prospects for Control. The text ends with a fine summary and closing.

There is an index of 14 pages, many tables and some line drawings, and two electron micrographs of HbSAg—one from plasma and one produced as a recombinant construction in yeast. The several lectures on AIDS are very good and include HTLV III testing.

This is a very scientific book and I believe would offer some difficulties for the practicing physician. I recommend, however, that a copy of this should be on the shelf of every blood bank and be read completely by every blood bank director. I also recommend a complete reading by residents in clinical pathology during their time allotted to blood bank training.

Hugh F. Luddecke, M.D.

Neuro-Ophthalmology: Clinical Signs and Symptoms, 2nd Edition

Thomas J. Walsh, M.D., (ed). Philadelphia, PA, Lea & Febiger, 1985. Pp. 499. Illustrated. ($47.50)

This work is a truly valuable addition to the general ophthalmologist's library. The organization of the book is based on signs and symptomatology so that ready and rapid reference to diagnosis and procedures can be accomplished.

The book is well written and contains a surprising wealth of valuable information. There are excellent photographs, illustrations, charts, and diagrams—all of which make it the kind of text for quick reference.

There is emphasis on thorough history and examination and avoidance of any unnecessary and costly procedures. The sections on radiology and CT scanning are particularly interesting and well done. Causes of blurred vision are very well presented; the mention of Moya-Moya disease, originally described in Japan, is included. Visual field defects and their bases are presented.

I am grateful to the author and his contributors for compiling such a valuable addition to the neuro-ophthalmology literature.

S. Jerome Greenfield, M.D.

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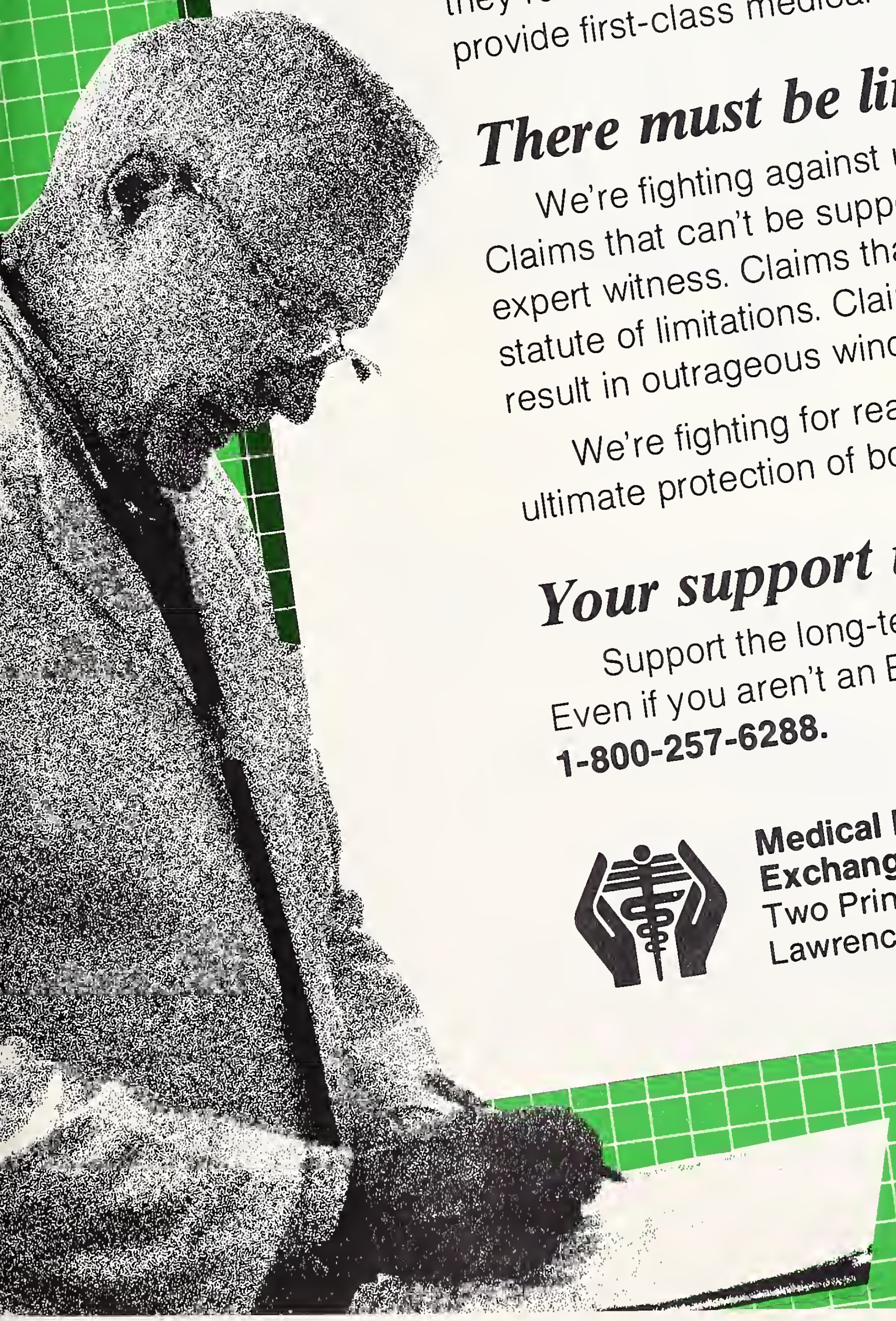

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OBITUARIES

Drs. Addeo; Andreson; Asbell; Booth; DeSandre; Gerne; Grundfest; Howeth; Marks; Pontilena; Rampona; Ruegsegger; Witmer

Dr. Anthony J. Addeo

Anthony John Addeo, M.D., died on January 13, 1986, at Wayne General Hospital, at the untimely age of 43. Born in Jersey City, Dr. Addeo received his medical degree from the University of Bologna, Italy, in 1971. He had received Brooklyn Jewish Hospital's Intern of the Year Award in 1972. An internist and nephrologist, he was a member of our Passaic County component, with board certification in internal medicine. Dr. Addeo had been affiliated with Wayne General Hospital, was an instructor in medicine there, had been chairman of their intensive care and cardiac unit, and had chaired the hospital's pharmacy committee.

Dr. Paul S. Andreson

A retired obstetrician and gynecologist, Paul Selby Andreson, M.D., died on September 14, 1985. Born in Sylvan Grove, Kansas, in 1912, Dr. Andreson was graduated from the University of Kansas School of Medicine in 1939, and after practicing in the states of Kansas and New York, he established an office in Elizabeth in 1961. He was a member of our Hudson County component and of the American Medical Association. Dr. Andreson was board certified in his specialty, and was a Fellow of the American College of Surgeons and of the American College of Obstetricians and Gynecologists. He had been affiliated with St. Elizabeth Hospital, Elizabeth. He served in the medical corps of the United States Army during World War II, he received the Bronze Star, and at his retirement from active service in 1961, held the rank of colonel.

Dr. Nathan Asbell

A surgeon who served the Cherry Hill and Camden communities, and was highly respected by his colleagues, Nathan Asbell, M.D., died January 24, 1986, at the age of 77. A native of Camden, Dr. Asbell received his medical degree from the University of London Faculty of Medicine (England) in 1934. He was a member of our Camden County component and of the American Medical Association. Dr. Asbell was a Fellow of the American College of Surgeons, of the Royal College of Surgeons, of the American Society of Abdominal Surgeons, and of the American Geriatric Society. His hospital affiliations were many, including the Skin and Cancer Hospital of Philadelphia; St. Joseph's Hospital, Philadelphia; medical director at Camden County General Hospital, Lakeland; and senior surgeon at West Jersey Hospital, Camden, since 1955. Dr. Asbell was active in county and state Society affairs, and had served on the former's legislative committee and the latter's Council on Legislation for many years. He also had been involved in community affairs; was physician for the city and county of Camden, served on boards of the American Cancer Society, the Mental Health Association, and Camden County United Way. Dr. Asbell was a recipient of MSNJ's Golden Merit Award in 1984, honoring his 50 years in medicine.

Dr. Walter S. Booth

An obstetrician and gynecologist who had served the community of Elizabeth for over 50 years, Walter Stephen Booth, M.D., died in Elizabeth General Medical Center, on January 17, 1986, at the venerable age of 81. A native of Elizabeth, Dr. Booth was graduated from Boston University School of Medicine in 1932 and immediately became a member of our Union County component and of the American Medical Association. He had been affiliated with St. Elizabeth's Hospital, Elizabeth, and the Elizabeth General Medical Center, and was a Fellow of the American College of Obstetricians and Gynecologists. During World War II, Dr. Booth served in the medical corps of the United States Navy in the Pacific theater, emerging with the rank of lieutenant commander. In 1982, he received MSNJ's Golden Merit Award, in recognition of his 50 years as a physician.

Dr. Louis A. DeSandre

Louis Adolph DeSandre, M.D., a family practitioner in Trenton, died at only 53 years of age in St. Lawrence Rehabilitation Center, Lawrenceville, on January 18, 1986. A native of Trenton, Dr. DeSandre was graduated from the University of Guadalajara, Mexico, in 1973. Prior to that he had been graduated from the Philadelphia College of Pharmacy and Science. Dr. DeSandre was a member of our Mercer County component and of the American Medical Association.

Dr. Timothy A. Gerne

A surgeon in Jersey City for over 50 years, Timothy Aloysius Gerne, M.D., died at his home in Morristown on December 1, 1985. Born in 1902, Dr. Gerne received his medical degree from New York Homeopathic and Flower Hospital in 1928. He was a member of our Hudson County component, and had been affiliated with Christ Hospital and the Medical Center both in Jersey City, and St. Mary Hospital, Hoboken. Dr. Gerne was a school physician in Jersey City for 35 years. During World War II, he served in the medical corps of the Army of the United States, attaining the rank of major. Dr. Gerne was a Fellow of the International College of Surgeons. In 1978, he was a recipient of MSNJ's Golden Merit Award in recognition of his 50 years as a physician.

Dr. Philip Grundfest

We just have been informed of the death, on June 13, 1985, of Philip Grundfest, M.D., a Kearny obstetrician and gynecologist. Born in 1915, Dr. Grundfest received his medical degree at the University of Arkansas School of Medicine in 1942. He was a member of our Essex County component and of the American Medical Association. Dr. Grundfest was affiliated with Clara Maass Memorial Hospital, Belleville; West Hudson Hospital, Kearny; and Newark Beth Israel Medical Center. During World War II, he served in the medical corps of the United States Army, with the rank of captain.

Dr. John L. Howeth

We have learned of the death in November 1985 of John L. Howeth, M.D., a retired general practitioner and orthopedic surgeon, at the age of 77. Born in Jersey City, Dr. Howeth received his medical degree from Columbia University College of Physicians and Surgeons, New York, in 1932. He established his practice in his native city, and became a member of our Hudson County component and of the American Medical Association. Dr. Howeth had been affiliated with Christ Hospital and the Medical Center, both in Jersey City. During the height of the polio epidemics he worked at the National Polio Foundation and the Elizabeth Kenny Institute, Jersey City. Dr. Howeth served in the medical corps of the United States Navy during World War II, emerging with the rank of commander. In 1982, MSNJ bestowed its Golden Merit Award on Dr. Howeth, honoring his 50 years as a physician.

Dr. Zelda I. Marks (Rosenthal)

A retired gynecologist and endocrinologist, Zelda I. Marks (Rosenthal), M.D., died on December 27, 1985, at Grace Plaza Nursing Home, Great Neck, New York, at the age of 78. Born on the Fourth of July, in New York City, Dr. Marks was graduated from Long Island College of Medicine, Brooklyn, in 1929. She had been on the staff at Newark Beth Israel Medical Center for nearly 50 years, and was chief of the endocrine laboratory there. Dr. Marks was a member of our Essex County component and of the American Medical Association. In 1980, she was recipient of the Elizabeth Blackwell Award of the American Medical Women's Association, of which she was a past president. In 1979, Dr. Marks received MSNJ's Golden Merit Award in recognition of her 50 years as a physician.

Dr. Nicholas D. Pontilena

An otolaryngologist, Nicholas Dominic Pontilena, M.D., died on January 4, 1986, in Hackensack Medical Center, at the untimely age of 37. Born in New York City, Dr. Pontilena received his medical degree from New Jersey Medical School (UMDNJ) in 1973. He was a member of our Bergen County component, and had been affiliated with Columbia-Presbyterian Medical Center, New York City, and Hackensack Medical Center.

Dr. Louis E. Rampona

Louis Edwards Rampona, M.D., a Princeton ophthalmologist from 1955 until his retirement in 1981, died on January 3, 1986, at his home. Born in Youngstown, Ohio, Dr. Rampona received his medical degree from Rush Medical College, Chicago, in 1934. For the first 20 years of his career, he practiced in Chicago, coming to Princeton in 1955. He became a member of our Mercer County component and of the American Medical Association. Dr. Rampona had been affiliated with The Medical Center at Princeton, and the New York Eye and Ear Infirmary, New York City. During World War II, he served in the medical corps of the Army of the United States, emerging with the rank of captain. In 1984, Dr. Rampona received MSNJ's Golden Merit Award, honoring his 50 years in medicine.

Dr. James M. Ruegsegger

A Montvale internist prior to his retirement, James Moore Ruegsegger, M.D., died in Pascack Valley Hospital, Westwood, on November 28, 1985. Born in Baltic, Ohio, in 1903, Dr. Ruegsegger received his medical degree from Western Reserve University School of Medicine, Cleveland, in 1931. He served as one of the medical consultants to President Franklin Delano Roosevelt, and also was appointed by Secretary of War Stimson to study outbreaks of pneumonia at military installations. Dr. Ruegsegger spent three years in the United States Navy's medical corps in China, Japan, and Okinawa. Upon his return to civilian life he was made director of clinical pharmacology at Lederle Laboratories. Dr. Ruegsegger was a member of our Bergen County component and of the American Medical Association, and was board certified in his specialty. In 1981, MSNJ bestowed on Dr. Ruegsegger its Golden Merit Award.

Dr. Ralph Witmer, Jr.

A Summit obstetrician and gynecologist, Ralph Witmer, Jr., M.D., died in 1985, at the age of 60. A native of Williamsport, Pennsylvania, Dr. Witmer was graduated from the University of Pennsylvania School of Medicine in 1952. He was a member of our Union County component and of the American Medical Association. Dr. Witmer was board certified in his specialty and was a Fellow of the American College of Obstetricians and Gynecologists. He had been affiliated with Overlook Hospital, Summit.

AUTHOR INFORMATION

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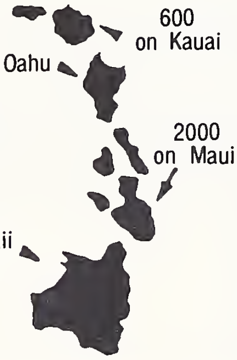

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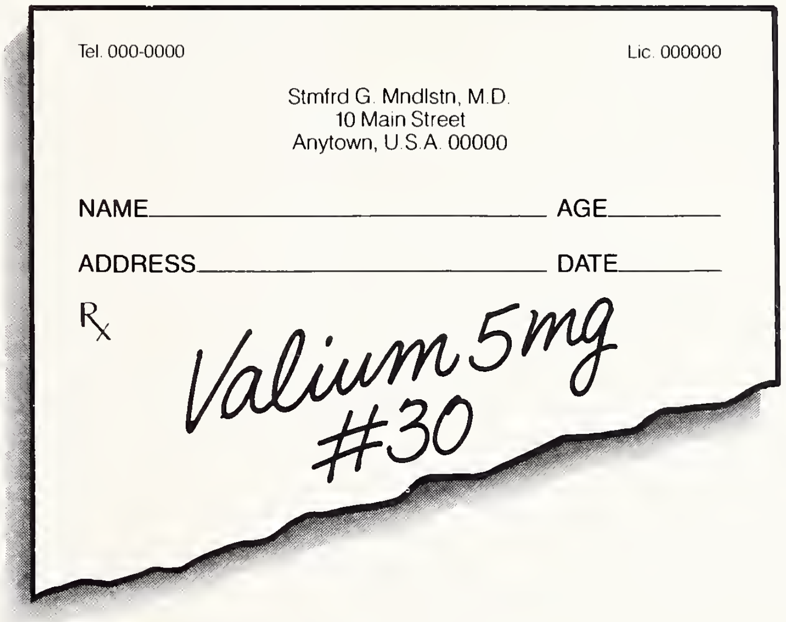

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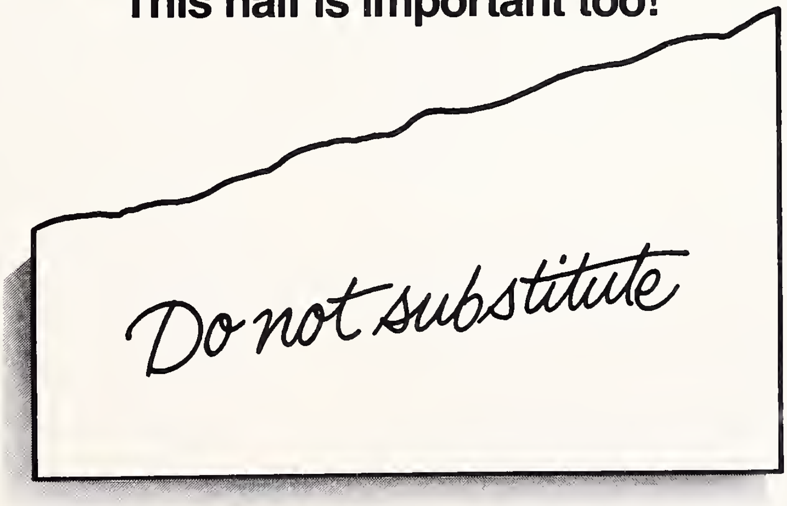

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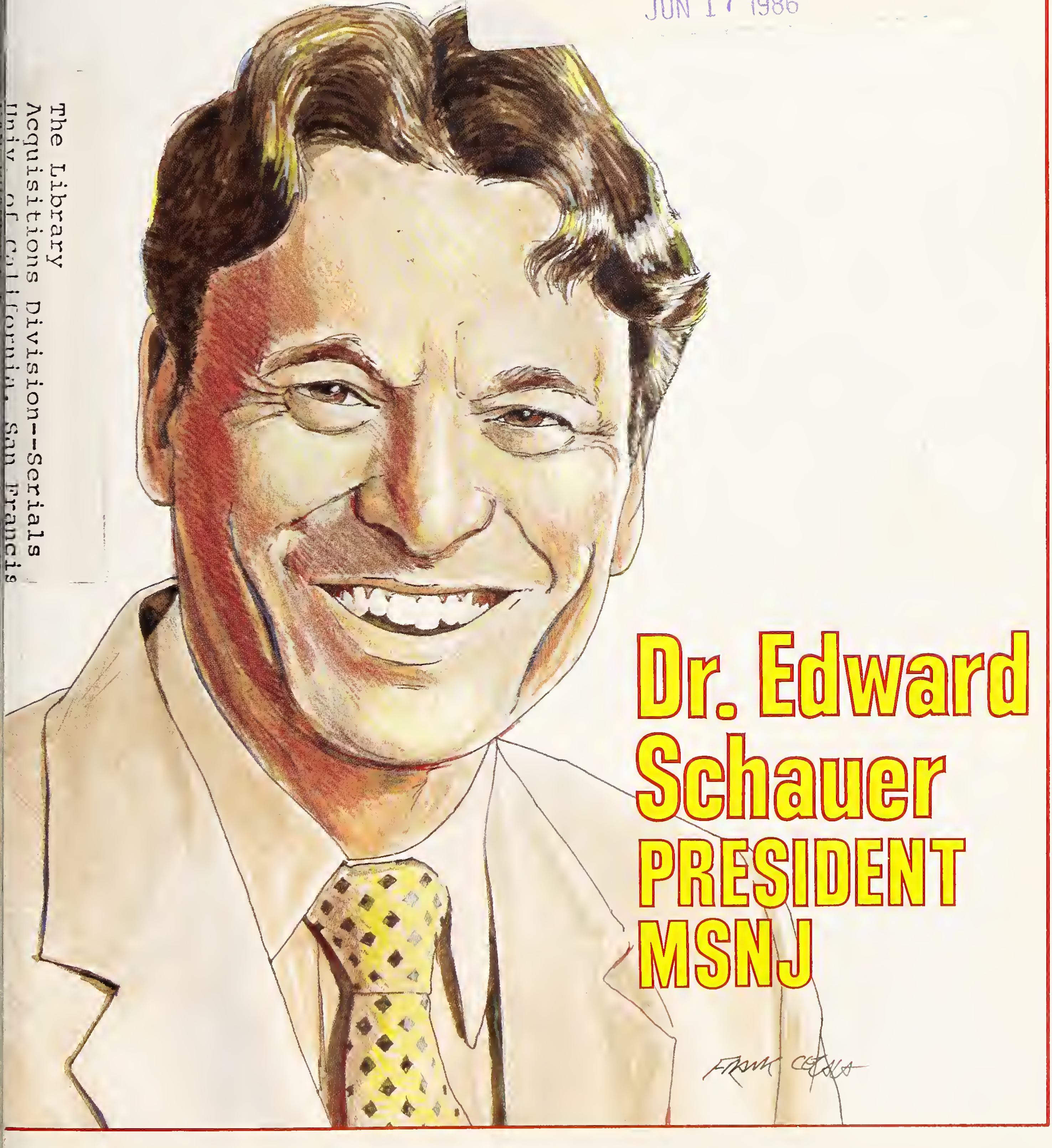

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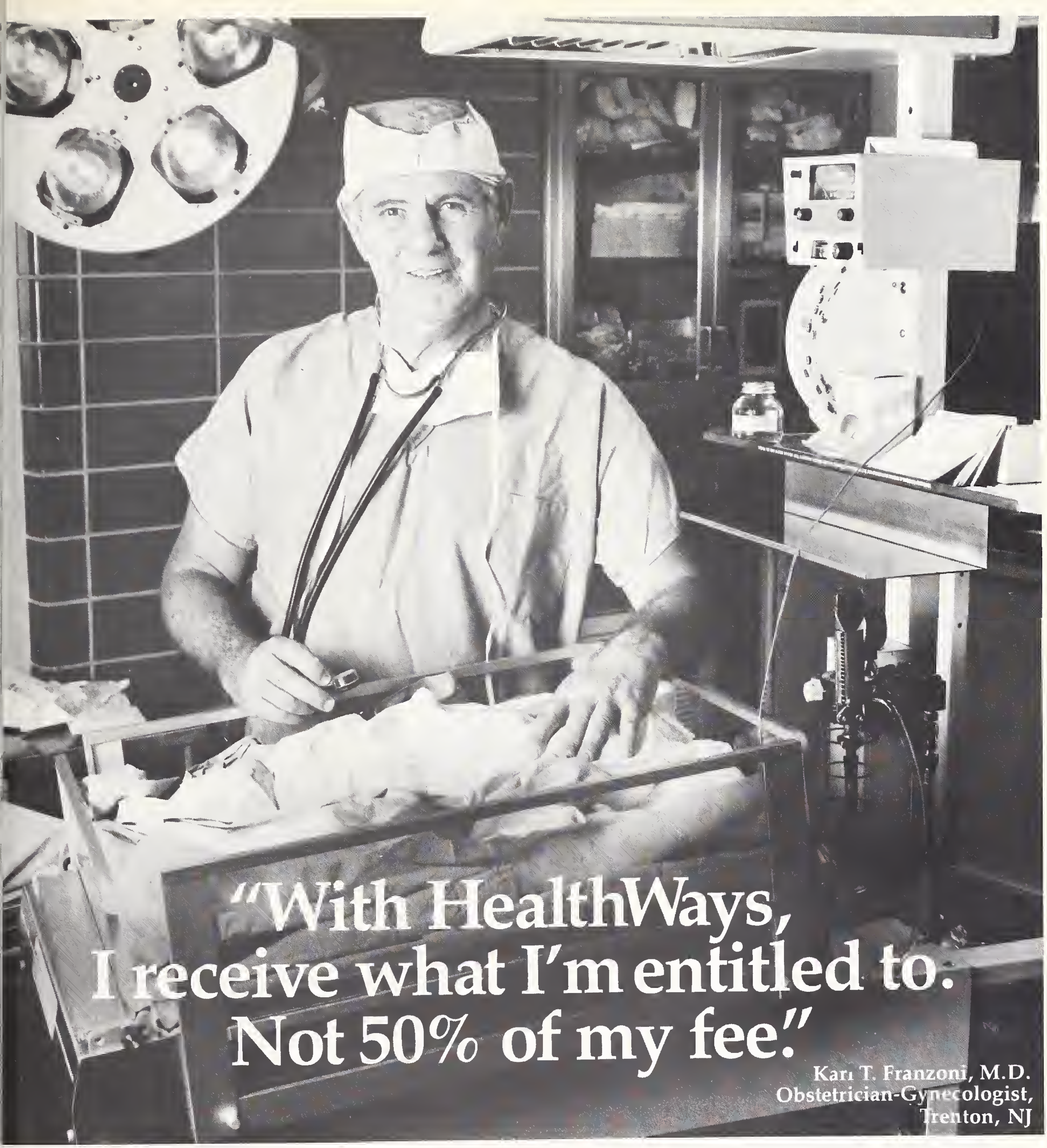
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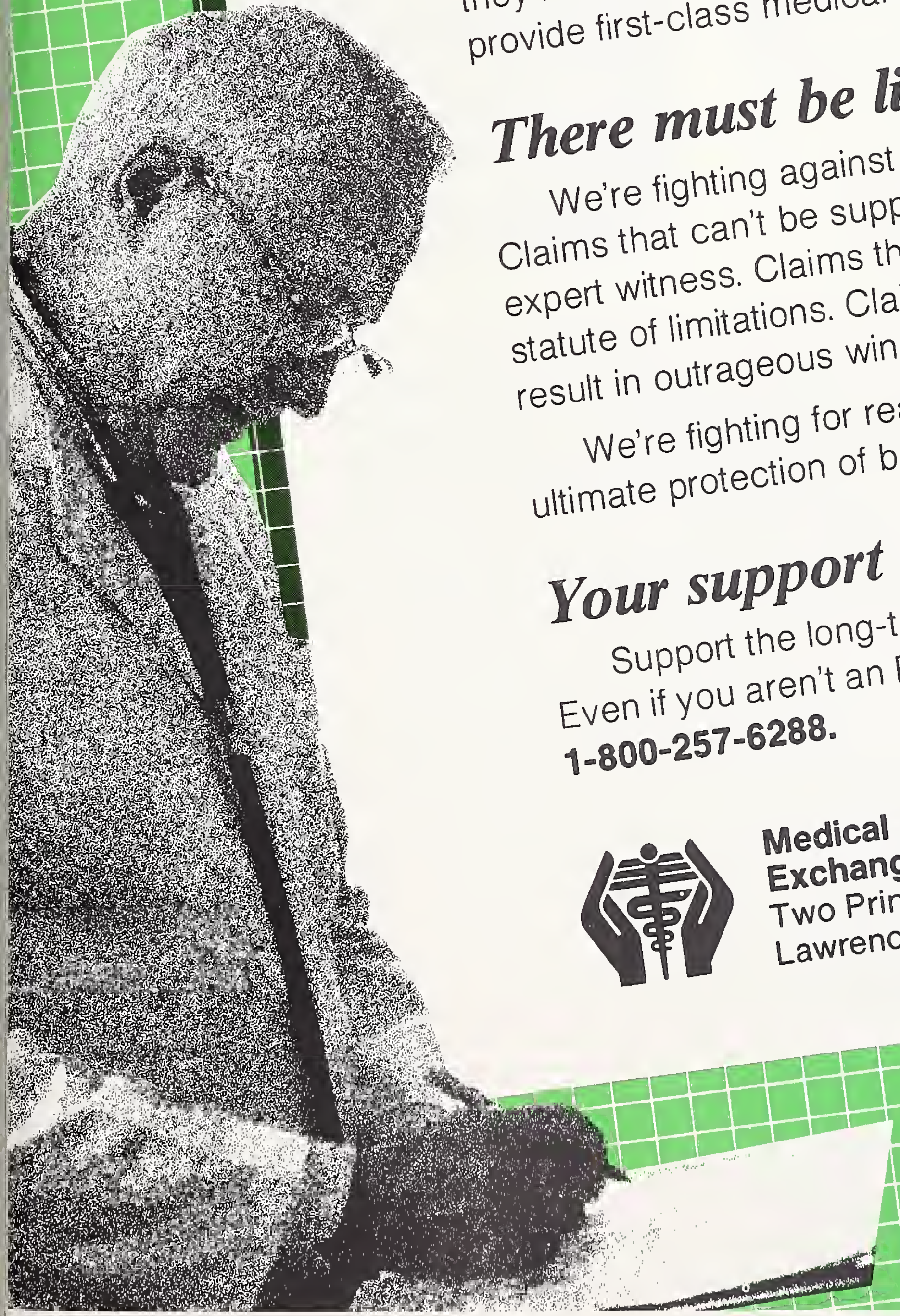
On The Cover: Edward A. Schauer, M.D., was installed as the 194th President of the Medical Society of New Jersey. Cover illustration by Frank Cecala.



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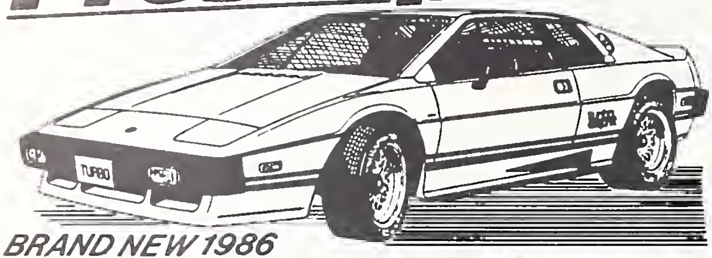

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MEMBERSHIP NEWSLETTER

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THE MEDICAL SOCIETY OF NEW JERSEY VOLUME 33

MEDICARE CHANGES RELEASED ON PHYSICIAN FEES

You all have received information from Prudential regarding the medical fee freeze under Medicare. Listed below are significant features of the new law. If you have questions, please call either the carrier or MSNJ.

1. Freeze on Fee Increases by Nonparticipating Physicians. Physicians who choose not to participate during the period May 1, 1986, through December 31, 1986, may not charge more for services rendered during that period than the actual charge level in effect during the quarter ending June 30, 1984. Physicians who willfully violate this provision may be subject to sanctions such as exclusion from Medicare participation and civil money penalties. This is the case regardless of the physician's earlier participation status. Under the recent statute, the restriction on fee increases by nonparticipating physicians is not extended beyond December 31, 1986.

2. Summary of Provisions for Remainder of 1986. In summary, the updated screen and fee freeze provisions apply as shown on the Table.

3. Special Rule for Hospital-Compensated Physicians. There is a special provision applicable to pathologists, radiologists, and other physicians who formerly were compensated by hospitals for furnishing physicians' services and who terminated those compensation arrangements after October 31, 1982.

4. Provisions in Effect in Calendar Years Beginning January 1, 1987. The recent legislation changes the cycle for updating reasonable charge screens and enrolling physicians and suppliers in the Medicare participation program. Beginning with 1987, reasonable charge screen updates and participation agreements will be effective for a 12-month period beginning January 1 of each year. There will be neither an update nor a participation/termination opportunity effective October 1, 1986. Also, the actual charges used to compute screens will be those in the period July 1 through June 30 rather than April 1 through March 31.

Effective with services rendered on or after January 1, 1987, the customary charges of both participating and nonparticipating physicians will be updated. However, with regard to prevailing charges, there will be a differential between participating and nonparticipating physicians. The prevailing charge applicable to the services of nonparticipating physicians will not be higher than the prevailing charge which was applicable in the previous year to physicians who were participating in that previous year. Thus, if you are a nonparticipating physician in the year beginning January 1, 1988, the prevailing charge applicable to your services will not exceed the prevailing charge in effect for participating physicians during 1987. If you elect to participate for the year beginning January 1, 1989, you will receive a full customary and prevailing charge update.

The recently enacted provisions also would establish a continual lag in the adjusted prevailing charge for only those physicians who choose to not participate during the May 1, 1986, through December 31, 1986, period. It does this by providing that the Medicare Economic Index (MEI) adjustment for the May 1, 1986, through December 31, 1986, period would be forever excluded from later adjusted prevailing charges applicable to those physicians, even if they are participating in those subsequent periods. However, key members of Congress have informed the Health Care Financing Administration that this continued lag was not intended by the Congress and that they plan to delete or modify this provision through a technical amendment at the earliest possible moment.

TABLE

Participation Status */		**Prevailing**	**Customary**	**Increases in Actual Charges**
Oct. 1, 1984-Sept. 30, 1985	**May 1, 1986-Dec. 31, 1986**			
Participating	Participating	Update	Update	Permitted
Nonparticipating	Participating	Update	Update	Permitted
Participating	Nonparticipating	No Update	Update	Not Permitted
Nonparticipating	Nonparticipating	No Update	No Update	Not Permitted

*/Under the new provisions of the law, participation status during the period from October 1, 1985, to April 30, 1986, does not affect whether or not customary and/or prevailing charges are updated.

PEOPLE'S MEDICAL SOCIETY

Here is a letter we received from the AMA.

"Thank you for sending us the mailing of the People's Medical Society. This material has been sent by them for a number of months, and copies have been sent to us at the American Medical Association since the spring of last year. We, too, have been outraged by this deception and false letter.

"We have done a number of things in response to the letter, including sending through "FED-NET" and the mail, a form letter and sample editorial or op-ed page reply that might be used by medical societies in their communities to attempt to correct the false information disseminated by the People's Medical Society. The *American Medical News* carried an editorial in its August 2, 1985, issue about the mailing.

"Unfortunately, in this land where we value our freedom of speech, we cannot force any individual or group to stop saying what they want. We can only continue our efforts to correct the misinformation that is being spread.

"We appreciate your calling the mailing to our attention. We did not know that it was continuing at this late date.

"Please be assured that we shall continue our efforts to see that the public is correctly informed about the AMA and what it is doing to assure high quality medical care for the nation's citizens."

GOALS OF THE FDA ACTION PLAN

New Drug Review—Make significant improvements in the new drug review process for human drugs, with the aim of further accelerating the review process while improving the quality. Specific activities and actions include: implementing the IND/NDA regulations; developing new models for conducting NDA reviews; reducing the backlog of supplemental NDAs; enhancing the use of automated data systems; and fully implementing the new program for post-1962 NDAs.

Medical Devices—Prepare specific recommendations to respond effectively to the legitimate criticisms that have been made about the Agency's implementation of the Medical Device Amendments of 1976. Special attention will be directed to those issues relating to bringing new products to market: premarket notifications [510(k)s]; premarket approval (PMA) applications; preamendments PMAs; and investigational device exemptions (IDEs).

Adverse Drug/Device Reporting—Improve the reporting of adverse drug reactions by increasing the number of reports from doctors and other health professionals submitted to FDA. Also, monitor the implementation of the new mandatory device reporting (MDR) systems, and develop improved reporting requirements for animal drugs and new requirements for biological products.

Risk Assessment—Direct the Agency's National Center for Toxicological Research to examine the assumptions underlying risk assessment. A basic question is how to extrapolate to humans the results of animal tests in which large doses of chemicals are given to small animals for their entire lifetime.

Risk Management—Develop quicker and more effective ways to remove or minimize a risk once it has been identified using early analysis options, full range of regulatory tools (education, enforcement, regulation, and voluntary compliance), organization of FDA's field operations, and coordination with federal, state, and local agencies.

SMOKING BAN IN MSNJ BUILDING

In view of the overwhelming evidence against smoking, developing liability laws, state laws related to the workplace, and financial considerations, we will be implementing a no-smoking policy for MSNJ employees within the headquarters. This same policy will apply to all members and guests.

COMMUNICATIONS WORKSHOP SPEAKERS

Videos of two noted speakers from the AMA's National Communications Workshop are available on loan to medical societies.

The opening remarks of John H. Dawson, M.D., AMA Trustee, titled, "The Physician's Image: It Begins in the Office," are available on a 51-minute tape.

Both speeches are being offered in ¾ inch and ½ inch formats and will be loaned for a period of two weeks. To reserve a copy of either presentation, contact: Carolyn Boozer, Department of Consulting Services at (312) 645-4416.

COMPLETING A DEATH CERTIFICATE

The funeral director in charge of the final disposition of a dead body is responsible for the total execution of the death certificate. In an effort to obtain a neat, accurate, and legible death certificate in a timely manner, the State Registrar requests that you print in the extreme lower left-hand corner, to the left of the words cause of death, the date of death and name of decedent as known by the attending physician.

The funeral director, upon assuming control of the death certificate and in consultation with the next of kin, then would verify the proper spelling of the decedent's name. Item 1 and 2 on the death certificate would be completed by the funeral director. If a discrepancy became apparent, the funeral director would be responsible to notify the attending physician so that his/her records could be adjusted accordingly. It also is strongly recommended that the cause of death be printed or written in a legible manner. Said procedure should help reduce the amount of querries.

If you have any questions please feel free to call the State Registrar at 609-292-4087.

PREADMISSION CERTIFICATION

Most preadmission certification programs in New Jersey are not operating within state law. If you have had an adverse experience with one of these, please report it to us with all essential details documented. We have established a special mechanism with the enforcement division of the Insurance Department to resolve these matters. Write to: Vincent A. Maressa, Executive Director, Medical Society of New Jersey, Medical Society of New Jersey, Two Princess Road, Lawrenceville, NJ 08648.

FINI

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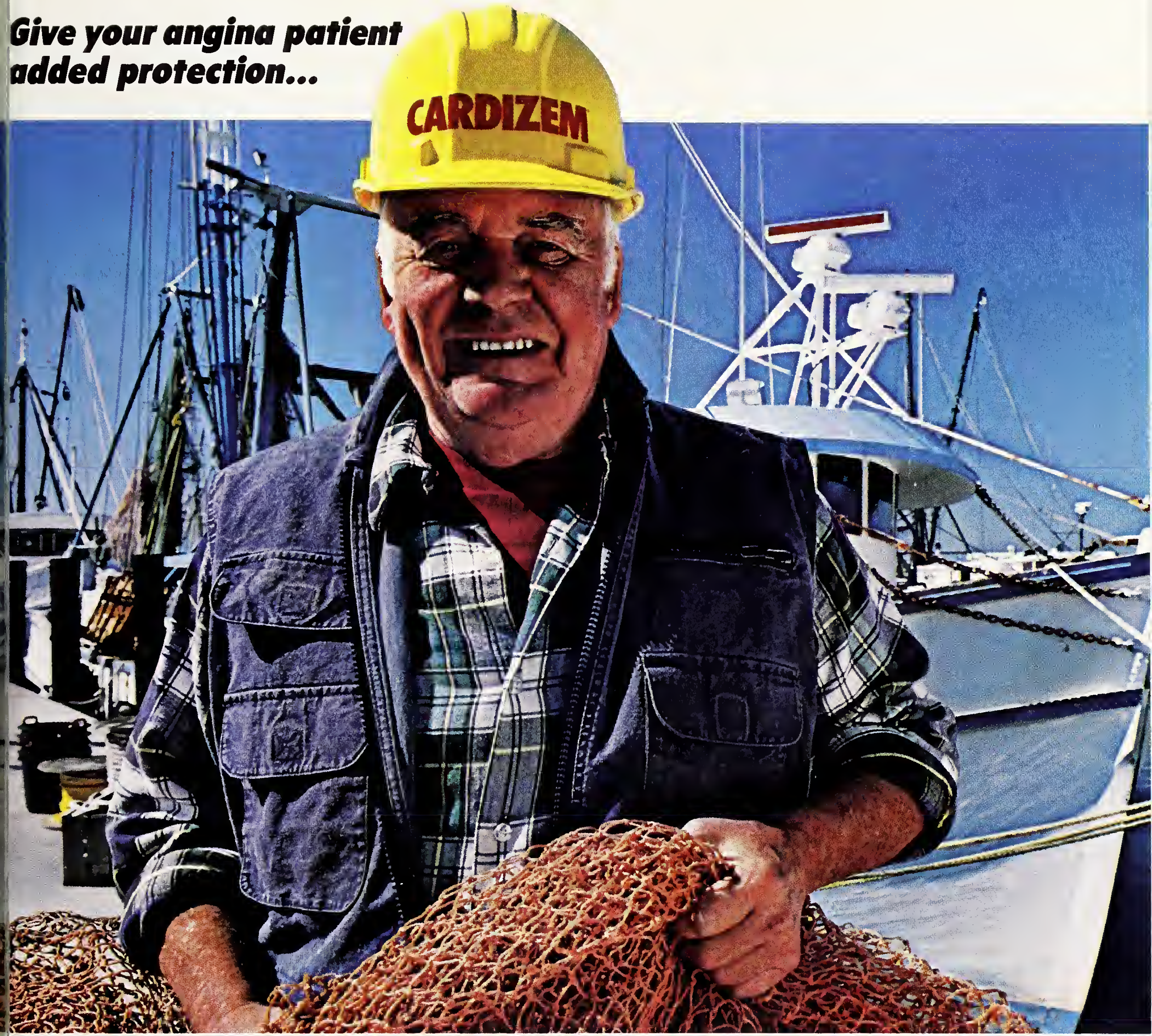

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- ***Compatible with both beta-blockers and nitrates[7]***

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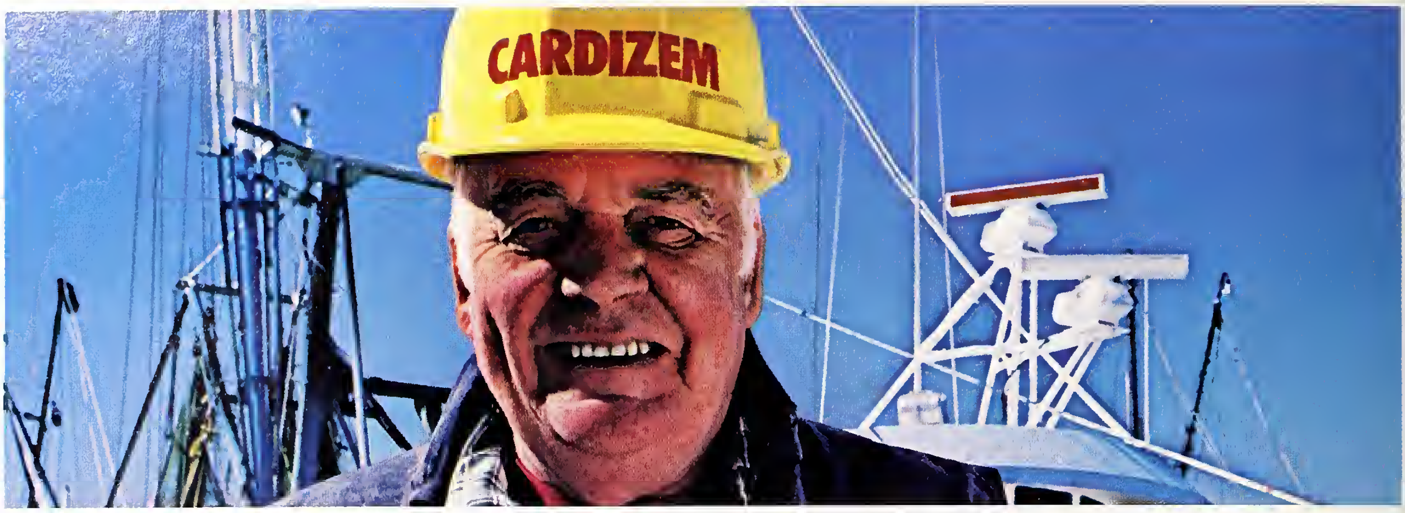

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FEWER SIDE EFFECTS IN ANTIANGINAL THERAPY

BRIEF SUMMARY

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist).

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm**. CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina)**. CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction**. CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure**. Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension**. Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury**. In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM (diltiazem hydrochloride) in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy** — CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

CARDIZEM 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other. Issued 4/1/84

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LEGISLATIVE UPDATE

Status of Legislation on Which MSNJ Has Taken an Active Position

Bill	Subject	MSNJ Position	Committee	Present Status
S-100 Bubba	Requires physicians to personally present and obtain surgical consent forms and signatures. The form shall state the name of any assisting physician that performs surgery under the supervision of the attending.	Active Opposition	Senate: LIP	In committee
S-124 Bubba	Requires express written consent to surgery except in cases of physical or mental incapacity or emergencies. The State Board, after consultation with the Health Department, the Medical Society, and the Hospital Association shall prescribe the consent forms which shall be used by physicians.	Active Support	Senate: LIP	Out of committee; 2nd reading
S-179 Brown	Requires the Department of Health to prepare a booklet on breast cancer which must be given to breast cancer patients by their physicians.	Active Opposition	Senate: LIP	In committee
S-281 Dumont	Provides for a three-year statute of limitations, except for fraud, intentional concealment, or nontherapeutic or diagnostic purpose. Minors have until age 11 on any injury prior to age 8.	Active Support	Senate: LIP	In committee
S-345 Ewing	Creates a commission to advise the Governor and the legislature regarding any proposals to regulate or license new professions.	Active Support	Senate: LIP	In committee
S-403 Rand	Grants immunity to peer review organizations when the organization is recognized by the Department of Health. Also provides for confidentiality of materials and records of the PRO.	Active Opposition	Senate: LIP	In committee
S-440 Garibaldi	Provides that cancers caused by exposure to heat, cold, radiation, or a known carcinogen which manifest themselves in active police and firemen are assumed to be accidental and work-related disabilities.	Active Opposition	Senate: SGF&IR&VA	In committee
S-492 Garibaldi	Requires a second physician to be present at any termination of pregnancy procedure if the attending has determined the unborn fetus is 24 weeks of age or older. The second physician is to provide immediate care to the child if it is born alive.	Active Opposition	Senate: IHW	In committee
S-493 Garibaldi	Requires the State Board of Medical Examiners to conduct a study as to when fetal viability occurs so that the conduct of physicians may be governed accordingly. Having conducted such a study, the Board is to promulgate guidelines regarding the termination of pregnancy.	Active Opposition	Senate: IHW	In committee

Bill	Subject	MSNJ Position	Committee	Present Status
S-539 Cardinale	Exempts the private practice of medicine from the certificate of need law.	Active Support	Senate: IHW	In committee
S-600 Bassano	Requires every physician attending a woman at the time of delivery, miscarriage, abortion, or during the prenatal period to perform an Rh test. If the test is Rh negative the physician shall, within 24 hours of receipt of the results, advise the woman of its significance and the availability of preventive treatment.	Active Opposition	Senate: IHW	Passed in Senate; in Assembly committee (HHR)
A-137 Muhler	Creates a 27-person Board within the Department of Human Services vested with the authority to release or continue the commitment of persons who were found incapable of standing trial or not guilty by reasons of insanity. A Superior Court judge currently makes these decisions.	Active Opposition	Assembly: HHR	In committee
A-181 Villane	This bill would establish and license two categories of social workers and would create a Board of Social Work Examiners in the Department of Law and Public Safety whose powers and duties, among others, would be to administer the act, examine and license candidates for the various categories of social work, and promulgate rules and regulations necessary for the effective enforcement of the act. The two categories of licensed social work would be 1) social work specialists, who would be required to have a doctorate in social work or a master's degree from an accredited school of social work and 2) social workers who would need a baccalaureate degree from an accredited college or university social work or social welfare program. The bill would "grandfather" in all persons currently in practice, provided they have been in practice in one of the two licensed categories for two of the last five years and apply to be licensed within 180 days from the effective date of this act.	Active Opposition	Assembly: HERP	In committee
A-200 Kosco	Creates a Commission to study the certificate of need process. The Commission would consist of 15 members. Two of the 15 would be physicians. Their report is to be submitted to the legislature and the Governor.	Active Support	Assembly: HHR	In committee

Senate Reference Committees
LIP: Labor, Industry, and Professions
SGF&IR&VA: State Government, Federal & Interstate Relations & Veterans Affairs
IHW: Institutions, Health & Welfare

Assembly Reference Committees
HHR: Health and Human Resources
HERP: Higher Education and Regulated Professions

Bill	Subject	MSNJ Position	Committee	Present Status
A-320 Zangari	Creates a separate Board of Chiropractic Examiners within the Division of Consumer Affairs.	Active Opposition	Assembly: HERP	In committee
A-360 Garvin	Requires the Department of Health and the State Board of Medical Examiners to prepare a booklet on all aspects of the treatment of breast cancer. Attending physicians are required to distribute the book to appropriate patients and to discuss it with them.	Active Opposition	Assembly: HHR	In committee
A-478 Muziani	Directs the State Board of Medical Examiners to review the issue of when fetal viability occurs and to provide guidelines to physicians regarding procedures to terminate pregnancy.	Active Opposition	Assembly: HHR	In committee
A-479 Muziani	Requires a second physician to be present whenever a physician terminates a pregnancy involving a live fetus of 24 weeks of age or older.	Active Opposition	Assembly: HHR	In committee
A-482 Muziani	Requires a physician to advise pregnant patients of the effects of alcohol and certain drugs on a fetus. The woman shall sign a form confirming the advice was given. The physician is to retain the original and provide the patient with a copy.	Active Opposition	Assembly: HHR	In committee

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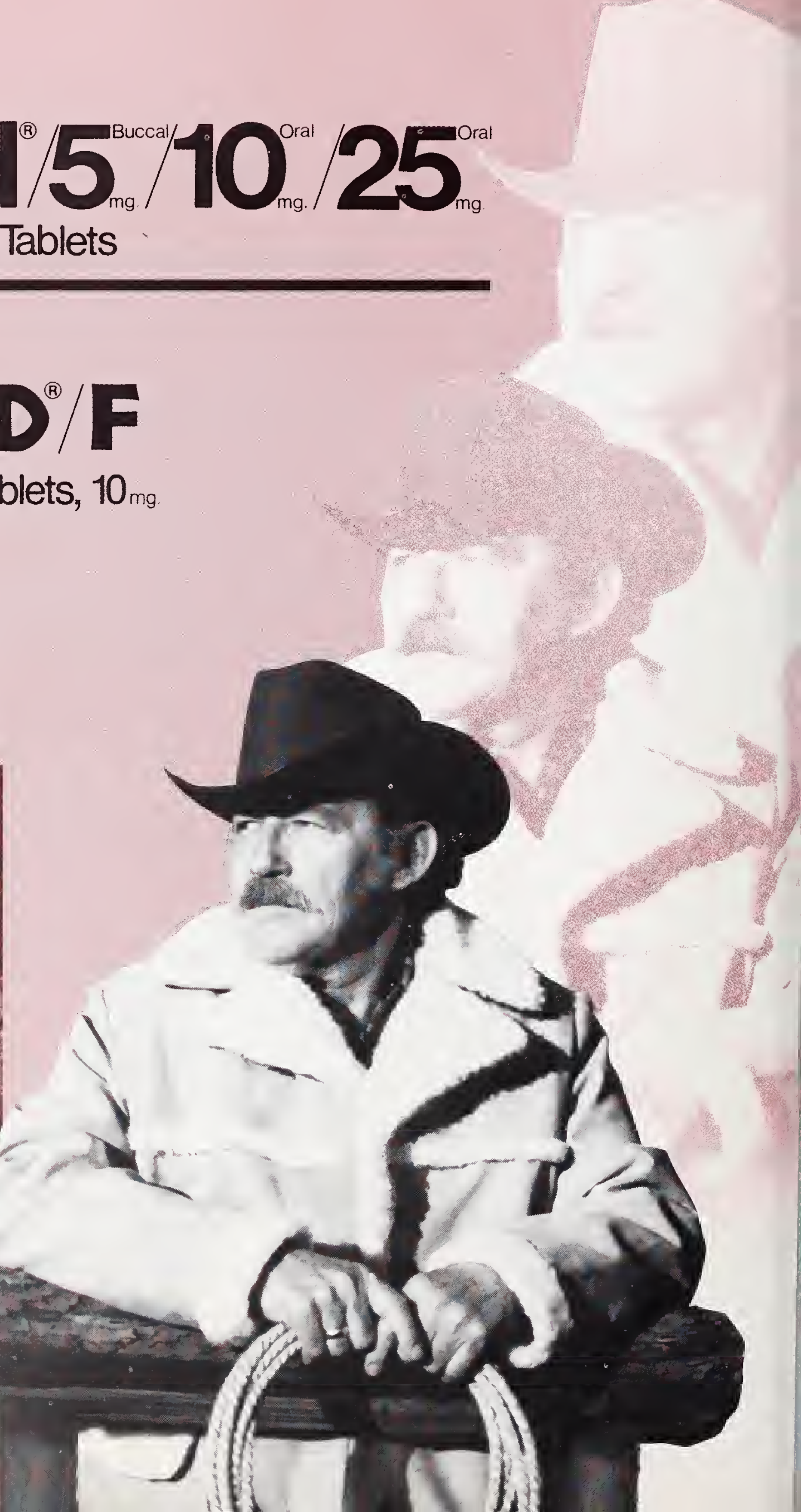

PROFESSIONAL LIABILITY COMMENTARY*

The Effects of Malpractice

Drug Side Effects; Impairment and Malpractice; AMA Malpractice Rules

The Illinois First District Appellate Court held that doctors, hospitals, and drug manufacturers have a nondelegable duty to warn users of the known adverse effects of prescription drugs and that they will be liable for injuries which occur to both users and third parties as a result of failure to warn. The case involved a patient under psychiatric treatment who was prescribed Prolixin Decanoate® and Thorazine®. The patient was injected with Prolixin Decanoate® and given Thorazine® orally. On the same day, he was discharged from the hospital. He then had an alcoholic drink, drove a car with the plaintiff as a passenger, and had an accident. The plaintiff suffered severe injuries and sued the drug manufacturers for failure to provide adequate warnings of the adverse effects of the drugs. The plaintiff also sued the psychiatrists and the hospital for failure to warn their patient that the drug would impair his mental and physical abilities.

The court stated that the existence of a legal duty is determined by: 1) the foreseeability that the defendant's acts or inactions will result in injury to the plaintiff; 2) the magnitude of the burden of guarding against the injury and the consequences of placing the burden upon the defendant; and 3) public policy considerations.

Under the foreseeability standard, the court held that it is not essential that the defendants should have foreseen the precise hazard and that a duty may exist to one who is unknown and remote in time and place. It further held that it was foreseeable that failure to warn of the adverse effects of the drugs could result in injury to the plaintiff or the public. The defendant attempted to argue that the plaintiff's consumption of an alcoholic beverage and his negligent driving constituted a superseding intervening cause, i.e. that the alcohol and the negligent driving, not the failure to warn, was the cause of the injury. The court maintained that because no warning had been given by the defendants, the alcohol and the poor driving were reasonably foreseeable, not superseding intervening causes, of the accident.

The defendants also attempted to argue that the burden of owing a duty to a general member of the public who was not their patient is an unjust burden because they would be liable to "an indeterminate class." The court did not agree with this argument, stating that "The imposition of a pertinent duty to warn that implicitly extends to cover the general public would not make doctors, hospitals, or drug manufacturers any more liable to an indeterminate class than are those in other fields and professions when a member of the general public is injured as a proximate cause of the wrongful act or inactions. . . . We, therefore, see no reason to discriminate in favor of doctors, hospitals, or drug manufacturers at the expense of innocent victims."

As for the public policy considerations, the defendants argued that if the duty was extended to cover the general public it would inhibit them from prescribing certain beneficial drugs, would open the door to endless suits by an infinite class of plaintiffs, and would make them strictly liable. The court said that these arguments "miss the point." According to the court, the public policy problem involves balancing the claims of those who have been injured as a result of medical negligence against the public's right not to have to pay needlessly inflated medical costs. The court maintained that imposing this duty would not inflate costs. In addition, the court stated that "the victims, not the wrongdoers, need protection. The wrongful acts must be curtailed, not the rights of the victims of the wrongful acts."

Although the hospital argued it should not be liable for the acts of its physicians who in this case were independent contractors, the court held that the hospital has a duty to warn that is independent of the physicians' duty. The court based its holding on its analysis that hospitals, in addition to physicians, do indeed provide treatment:

> The old conception that a hospital does not undertake to treat the patient no longer reflects reality. Plainly, hospitals do far more than merely furnish an edifice for treatment. Rather, they regularly employ on a salaried basis a staff of physicians, nurses, pharmacists, technicians, and medical administrative professionals, and, not insignificantly, the costs for these personnel are factored into the

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*This item, from the Department of Professional Liability Control, MSNJ, was prepared by James E. George, M.D., J.D., and A. Ronald Rouse, who are, respectively, Director of the Department and Director of Special Projects.

> amount that patients are charged by the hospital (Darling *v* Charleston Community Hospital (1965), 33 Ill.2d 326, 332, 211N.E. 2d 253, 257). Also, hospitals now advertise and promote the treatment and care that patients receive at their particular hospital (e.g. Chicago Sun-Times, June 30, 1986, Medical Centers-Special Pullout Section), and it is not uncommon for members of the general public to choose a particular hospital for treatment and care simply on the basis of the reputation of the hospital itself. It follows that hospitals must assume certain responsibilities for the care and treatment of their patients (Darling, 33 Ill.2d at 332, 211 N.E.2d at 257). Within this context, we conclude that before a patient is discharged from a hospital, the hospital has a duty to warn the patient of the adverse effects of drugs that were administered by the hospital if the hospital knows or should know that the drugs will impair the patient's mental or physical abilities, or will be potentially dangerous in combination with particular foods, beverages, or other drugs. A patient should not be discharged from a hospital under circumstances which make the patient unknowingly a potential danger to himself or herself, and the public-at-large, because of drugs administered by hospital personnel. Here, we should bear in mind that the patient is charged by the hospital for both the services of the hospital personnel who administered the drugs as well as for the drugs themselves and that the duty arises only when the hospital knows or should know of the adverse effects of the drugs.

As for the product liability claim, the hospital argued that it cannot be held strictly liable for failure to warn of the adverse effects of drugs, as the hospital is providing a service, not selling a product. The court held that this would be true for injurious results of treatment or danger to the patient created by the medical decision to administer the drugs. In the case, however, the issue was the unreasonable condition of the drugs themselves and since the hospital charges patients for the drugs which they dispense, the hospital was within the chain of distribution of a product and therefore potentially liable under a products liability theory.

The defendants have sought a rehearing and also are petitioning for reconsideration for leave to appeal to the Illinois Supreme Court. (*Occurrence*, a bimonthly publication of the Chicago Hospital Risk Pooling Program, Vol. 6, No. 2, November-December, 1985)

EXPERT LINKS IMPAIRMENT AND MALPRACTICE

A preliminary study indicates there is a definite connection between physician impairment and medical malpractice, a New Jersey expert in chemical addiction told a Florida Medical Foundation-sponsored workshop.

David I. Canavan, M.D., medical director of the Medical Society of New Jersey's Impaired Physicians Program, said his survey was based on the cases of 61 impaired physicians in his program. While the number of malpractice cases generated by this group appeared to be smaller when compared with nonimpaired physicians, the cost of settlement of each case was significantly higher, Dr. Canavan said.

"We are going to investigate this further," Dr. Canavan told participants in the Committee on Impaired Physicians' Sixth Workshop on Intervention with Impaired Physicians. "We hope to get a larger sample by cooperating with other states."

Dr. Canavan's program now in its fourth year, has a current annual operating budget of $237,000, most of which is provided by two medical malpractice insurance companies in New Jersey. The Medical Society of New Jersey and state veterinary and osteopathic organizations contribute the remainder. He quoted one malpractice insurance official by saying that if the program spares his company one settlement a year, the contribution is more than repaid. (*The FMA Today*, January 1986)

PANEL REJECTS AMA MALPRACTICE RULES

Intensifying a dispute between the nation's lawyers and the nation's doctors, a special committee of the American Bar Association (ABA) has recommended rejecting all the American Medical Association's (AMA) major proposals for restrictions on malpractice suits.

The committee's report, to be considered for adoption as ABA policy, reflects a sharp conflict between the legal and medical professions over what the AMA asserts is a "crisis" created by expensive medical malpractice litigation.

Contrary to the doctors' claims for "special treatment," the 44-page report said there is no justification for fundamental changes in the system of resolving allegations of medical negligence through lawsuits for monetary damages—just as other personal injuries, or torts, are resolved.

The report said that "the medical profession, in seeking changes in the tort law system, has shown a willingness to trade away the rights of individuals in the hope of easing a perceived burden on itself."

The lawyers' move could sharpen head-to-head lobbying between the two powerful professions as legislators consider AMA proposals, issued early last year, to limit damages and attorneys' fees in malpractice suits.

The AMA, which has 271,000 members nationwide, has argued that the rapidly rising costs associated with malpractice litigation are driving doctors from the profession and the "reform" of the system is imperative to bring malpractice insurance premiums under control.

Among other measures, the organization is pushing for limitations on punitive damages, on damages for pain and suffering, and on contingent fees, the practice whereby lawyers represent plaintiffs in return for a share of any damages won.

James S. Todd, M.D., the senior deputy executive vice president of the AMA, said having an ABA committee pass on proposals for changing the litigation system was "like sending the wolves to watch the chicken coop." Even lawyers who defend doctors in malpractice suits "earn their income by the same inequities that allow the plaintiffs' bar to earn their income," he said. (*Trentonian*, February 4, 1986)

EDITORIAL

EDWARD SCHAUER, M.D. 194TH PRESIDENT

Dr. Edward Schauer was installed as the 194th President of the Medical Society of New Jersey.

The Medical Society of New Jersey installed Edward Augustine Schauer, M.D., of Farmingdale, as its 194th President. Doctor Schauer, a charter Diplomate of the American Board of Family Practice, is in practice with his brother, Joseph William Schauer, Jr., M.D., and his nephew, Joseph W. Schauer, III, M.D.

Born in Morrisville, Pennsylvania, Doctor Schauer attended Morrisville High School, and received his Bachelor of Arts degree from Colgate University in Hamilton, New York. In 1949, Doctor Schauer was awarded his medical degree from Jefferson Medical College, Pennsylvania. After an internship at the U.S. Naval Hospital, Long Island, New York, Doctor Schauer served in the Korean War as a medical officer; upon discharge, he entered private medical practice.

Doctor Schauer has been affiliated with Jersey Shore Medical Center, Neptune, since 1952. He was instrumental in founding the family practice department, serving as its chairman for five years. Doctor Schauer was the hospital's chief of staff for three years (1981 to 1984).

Doctor Schauer has been active in medical association activities for many years:

- He is a member of the American Medical Association and is an alternate delegate to the AMA, serving since 1979.
- He served as President of the Monmouth County Medical Society in 1970, and has been a Monmouth County delegate to MSNJ since 1958.
- He was a founder and the first President of the Monmouth County Chapter of the New Jersey Academy of General Practice; he served on the Academy's Board of Directors for ten years, as President of the New Jersey Academy in 1967 and Chairman in 1968, and Speaker of the House since 1972.
- He was a charter fellow of the American Academy of Family Physicians as well as the New Jersey representative in the American Academy of Family Physicians' House of Delegates from 1963 to 1976. He served on the national board for three years, and as its Vice-President in 1979.
- He has served as President of the Monmouth County Heart Association and chairman of numerous fund drives.
- He is a member of the Medical Advisory Committee to the National Society to Prevent Blindness—New Jersey Division.

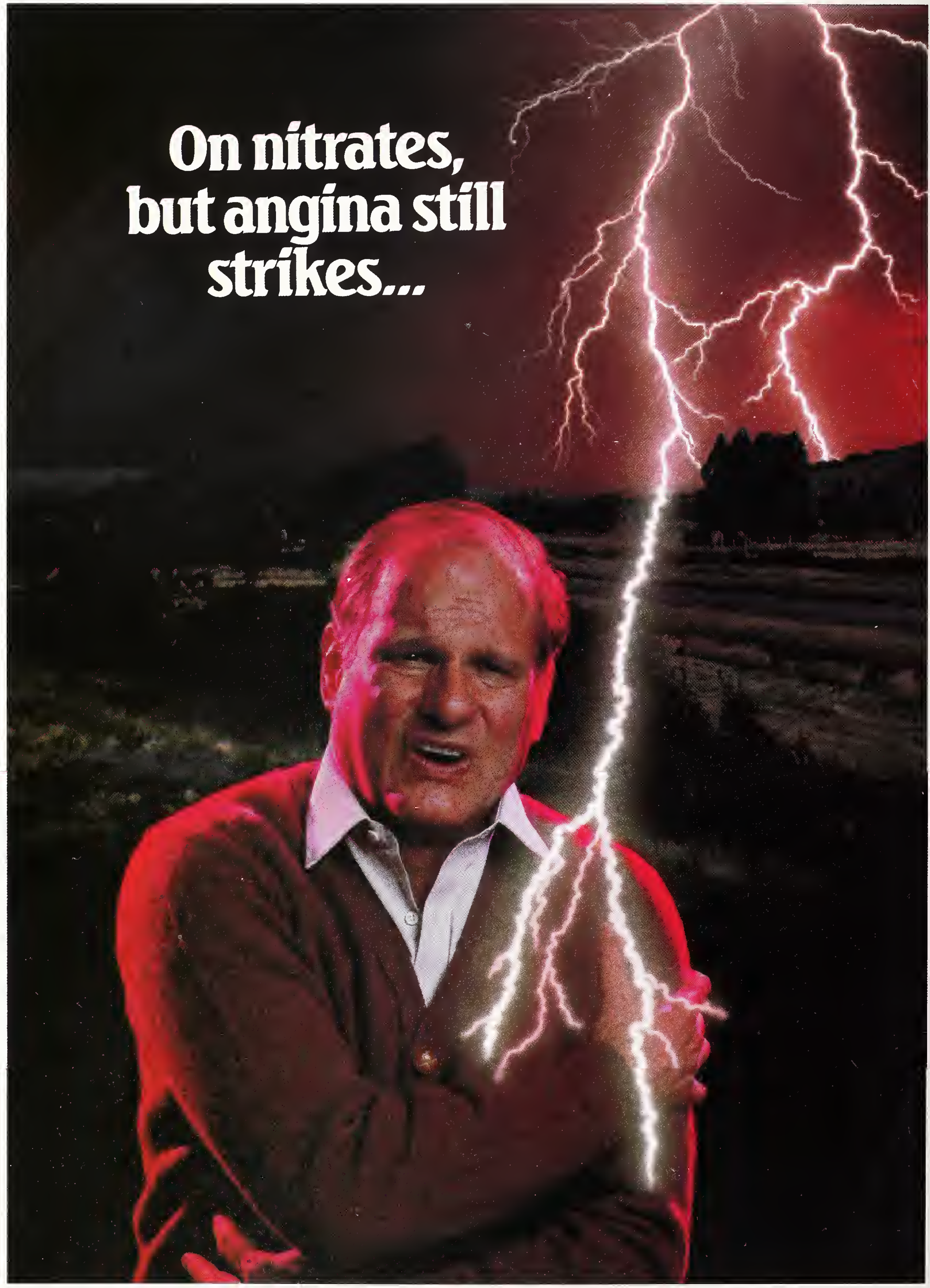
In his home community, Doctor Schauer is equally active:

- He is an associate member of the fire department.
- He was a member of the local school board, and has been school physician for eight Howell Township grammar schools since 1958.
- He is an active participant in the Pop Warner Football Program.
- He has been President of his local Holy Name Society.
- He is medical physician for Brisbane Child Treatment Center.

Doctor Schauer is married to the former Alice Walldov, a registered nurse and graduate of the Ann May School of Nursing and Monmouth College. Together they have four children: Alice Jean, Christine, Andrea, and Edward; and four grandchildren: Erik, Edward, Brian, and Barbara.

His wife enjoys reading and traveling. Doctor Schauer enjoys singing, volleyball, and politics.

The Medical Society of New Jersey congratulates Edward A. Schauer, M.D., and wishes him every success during his term as President.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Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Such elevations may disappear even with continued treatment; however, four cases of hepatocellular injury by verapamil have been proven by rechallenge. Periodic monitoring of liver function is prudent during verapamil therapy. Patients with atrial flutter or fibrillation and an accessory AV pathway (e.g. W-P-W or L-G-L syndromes) may develop increased antegrade conduction across the aberrant pathway bypassing the AV node, producing a very rapid ventricular response after receiving ISOPTIN (or digitalis). Treatment is usually D.C.-cardioversion, which has been used safely and effectively after ISOPTIN. Because of verapamil's effect on AV conduction and the SA node, 1° AV block and transient bradycardia may occur. High grade block, however, has been infrequently observed. Marked 1° or progressive 2° or 3° AV block requires a dosage reduction or, rarely, discontinuation and institution of appropriate therapy depending upon the clinical situation. Patients with hypertrophic cardiomyopathy (IHSS) received verapamil in doses up to 720 mg/day. It must be appreciated that this group of patients had a serious disease with a high mortality rate and that most were refractory or intolerant to propranolol. A variety of serious adverse effects were seen in this group of patients including sinus bradycardia, 2° AV block, sinus arrest, pulmonary edema and/or severe hypotension. Most adverse effects responded well to dose reduction and only rarely was verapamil discontinued. **Precautions:** ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacologic effects. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, close surveillance of vital signs and clinical status should be carried out. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patients should be carefully monitored to avoid over- or under-digitalization. ISOPTIN may have an additive effect on lowering blood pressure in patients receiving oral antihypertensive agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Clinical experience with the concomitant use of ISOPTIN and short- and long-acting nitrates suggest beneficial interaction without undesirable drug interactions. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. *Pregnancy Category C*: There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use. **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia: HR < 50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%), elevations of liver enzymes have been reported. (See *Warnings*.) The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: ecchymosis, bruising, gynecomastia, psychotic symptoms, confusion, paresthesia, insomnia, somnolence, equilibrium disorder, blurred vision, syncope, muscle cramp, shakiness, claudication, hair loss, macules, spotty menstruation. **How Supplied:** ISOPTIN (verapamil HCl) is supplied in round, scored, film-coated tablets containing either 80 mg or 120 mg of verapamil hydrochloride and embossed with "ISOPTIN 80" or "ISOPTIN 120" on one side and with "KNOLL" on the reverse side. Revised August, 1984. 2385

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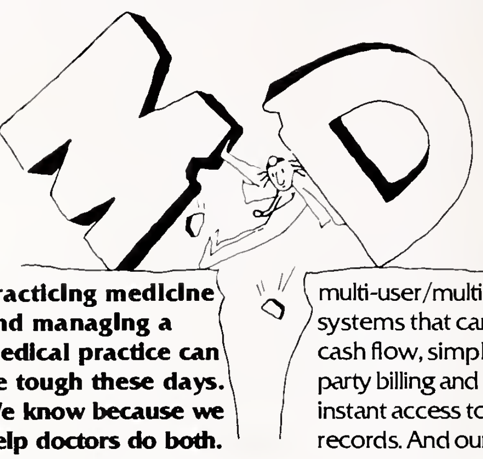

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INAUGURAL ADDRESS: MEDICINE TODAY

EDWARD A. SCHAUER, M.D., FARMINGDALE

Edward A. Schauer, M.D., presented this Address at his inaugural, on May 2, 1986, at the Annual Meeting of the Medical Society of New Jersey, at Americana Host Farm Resort, Lancaster, Pennsylvania.

As we embark on the 220th year of the Medical Society of New Jersey, it might be appropriate to take an accounting of our stewardship and reassess our progress and goals.

1. Membership. At the end of 1985, we had approximately 9,600 members. This is not a bad figure, but we could be doing better. Membership is the lifeblood of any organization and all of us must cultivate, nurture, and bring to fruition, a larger tally. We need new members—members with interest, ideas, concern, vision, courage, and strength to carry our banners ever forward. This is not an easy task; in fact, it has become a very, very difficult proposition. As physicians, and organized medicine as a whole, we want to practice the best possible medicine; at the same time, we want to be able to fend off the forays of those who would like to change the basic concepts of who will deliver medical care, how it should be delivered, and the mechanisms for remuneration. Yes, indeed, we need membership in quantity and of the most superb quality.

2. Education. Education must continue to be the cornerstone of medical practice. For many years, the Academy of Medicine of New Jersey and our Committee on Medical Education have worked hand-in-hand to produce a plethora of excellent programs for our physicians. These are given in all areas of our state throughout the calendar year. In addition, the specialties have their programs as do our medical schools. There is indeed ample opportunity for all New Jersey physicians to keep their educational acuity sharp.

3. Legislation. Each year legislation occupies a great deal of our time and effort because of activities in the legislative arena. There are increasing numbers of bills in both the state legislature and in Washington that are related directly to the practice of medicine that must be addressed. In addition, of course, we have other bills that have an indirect bearing on our profession. Finally, there still is a third group that deal with people in general—our patients.

We cannot afford to proceed with blinders on; nor can we use a faulty compass to chart our course in this legislative morass. As a medical society, we must be concerned, well informed, active, and alert.

Our Council on Legislation has been more active and, if possible, busier each year trying to evaluate and digest all the verbiage flowing from the State House. Over the past five years attendance at meetings and input into the discussions have been greater. This is encouraging but we must continue to stir the pot. We need thoughts, suggestions, and structured solutions.

4. Medical Service. Medical service has to do with the character of our practice of medicine, the standards we adhere to, and the impact of economic and social factors as they impinge on this caring for our patients. Under this format, the Medical Society of New Jersey must be concerned about all of our members—those in private practice, salaried practice, teaching, research, government service, administrative medicine, and pharmaceutical medicine. We must be concerned with the milieu in which our members practice—a solo private office, a multispecialty private office, an office or department within the hospital, the medical school, the emergency room, or an urgi- or emergicenter. And, of course, we must be concerned with the mechanism of remuneration for medical care: fee-for-service, third-party payment via usual, customary, and reasonable indemnity payment system, and capitation or relative value fee schedule.

5. Finances. How are we doing fiscally? We have a dedicated and efficient staff led by Mr. Maressa. They are at our beck and call, and attempt to project our best image. In addition, we have a very adequate and modern medical society building and it is being used not only for our purposes but for other medical groups. As the Committee on Finance and Budget reveals, we are in the black, balancing our budget, and trying to properly administer and foster all appropriate programs. However, we all must recognize and realize that if any of us, individually or collectively, wish a portion of organized medicine to do more, it involves additional money. We then must make the decision of the source of these funds, and ask if this involves raising dues.

LOOKING AHEAD

Do we have problems? Yes, we certainly do but then again that is not unusual for organized medicine including our own Medical Society of New Jersey.

I will touch upon just a few of our difficulties. One of our greatest problems is the medical liability crisis. The medical liability climate is extremely poor and does not seem to be improving. More physicians are being sued every year. Let me be perfectly clear: some liability suits are in order and should be properly adjudicated, but some are frivolous and others have brought about a distortion of the concept of liability. On the other side of the coin, the awarding of excessive and unpredictable damages and the high transaction costs for cases have driven premiums for insurance out of sight. As costs have gone up, physicians have had to pass some of this increase on to the patient and, in addition, practice more defensive medicine which costs more. It has been a rather vicious cycle. In addition, in some states it has become more difficult to obtain adequate insurance. However, there is a new development in this liability field—many other segments of society are beginning to have the same difficulties—schools, townships, counties, restaurants, businesses—they are beginning to perceive some of these concerns. At this time, we may be reaching the best consensus on this topic we ever have had. We must bring forth every effort to stabilize this liability picture. There are bills in the state legislature and in the national Congress (Hatch bill S 1804 and Lent bill HR 3865) that should be strongly supported and passed. To accomplish this will take phone calls, letters, appearances at hearings and meetings, political action, and money—no effort should be spared.

Another problem facing our physicians is the myriad of alternative medical delivery systems. They have almost become ubiquitous and are growing like the proverbial Topsy. They include HMOs, PROs, and IPAs; they are quite foreign and somewhat incomprehensible to many physicians who never have been involved in any arrangement other than private practice. Those who attended one of the three meetings on this topic presented this year under the wise guidance of Dr. Fioretti and excellent tutelage of Dr. Bernard Robins should be well informed. Those of you who did not have the opportunity, please review the literature carefully: know the background of any plan and who is running it; how it functions; who makes the decisions. Be very careful that you do not sacrifice some of your freedoms of choice and practice options for some so-called financial security.

We have many concerns with public health problems including alcoholism, drug abuse, suicide, and child molestation. More recently, AIDS has come to the fore and this condition is causing great anxiety and fear among large numbers of Americans. As of early this year, there were 16,000 cases in the United States and 1,000 of these in our own state. Because of the high fatality rate and many misconceptions about this disease, the gauntlet has been thrown to us. We must educate and inform the public of the truths and untruths of this disease in a reasonable and realistic way. This is not a time for panic—this is a time for reasoned judgment.

In spite of problems, as leaders of medicine we must be astute and mature enough to address them forthrightly and develop solutions that are in the best interests of our patients and yet do not interfere with the patient-doctor relationship nor mar the sacred trust and faith that the patient has for an individual physician.

For our patients I think we should stress preventive medicine and lifestyle in an attempt to promote good health and a high quality of life. Babies and children need their routine immunizations; adults need immunizations depending on their age, type of work, and exposure to disease. We should counsel our patients on diet—what types of foods are more nutritious, what foods perhaps should be avoided and what is a good balance including an intelligent, judicious use of vitamins. We should encourage some form of regular exercise; Americans have become more exercise conscious during the last 15 years and this is good. Smoking claims approximately 350,000 deaths in our country every year, plus many more thousands incapacitated due to many chronic conditions. We must continue to encourage and assist our smokers to stop, but, more importantly, we must educate our youth in order that they do not ever begin. Many medical groups including the New Jersey Divisions of the American Cancer Society, American Heart Association, American Academy of Family Physicians, and American College of Preventive Medicine have been promoting antismoking programs for years and we should reinforce

We should stress preventive medicine and lifestyle in an attempt to promote good health and a high quality of life.

and complement these. Burlington County Medical Society developed and presented an excellent film entitled "Smoking: Friend or Foe?" This film or comparable ones should be widely disseminated throughout our school system, particularly in the lower grades. The AMA has a new goal of having a smokeless society by the year 2000—the Medical Society of New Jersey should be one of the heralds for this new crusade.

As a corollary to smoking, we also must strongly discourage the use of illict and controlled substance drugs. These entities lead to horrible dependencies and death. In addition, in our state, they are the leading transmission cause of AIDS. We must promote the necessary education of all our citizens but particularly our youth.

And, finally, in our general advice about quality of life we should remember that if one must drink alcohol, do it in moderation and keep it under control.

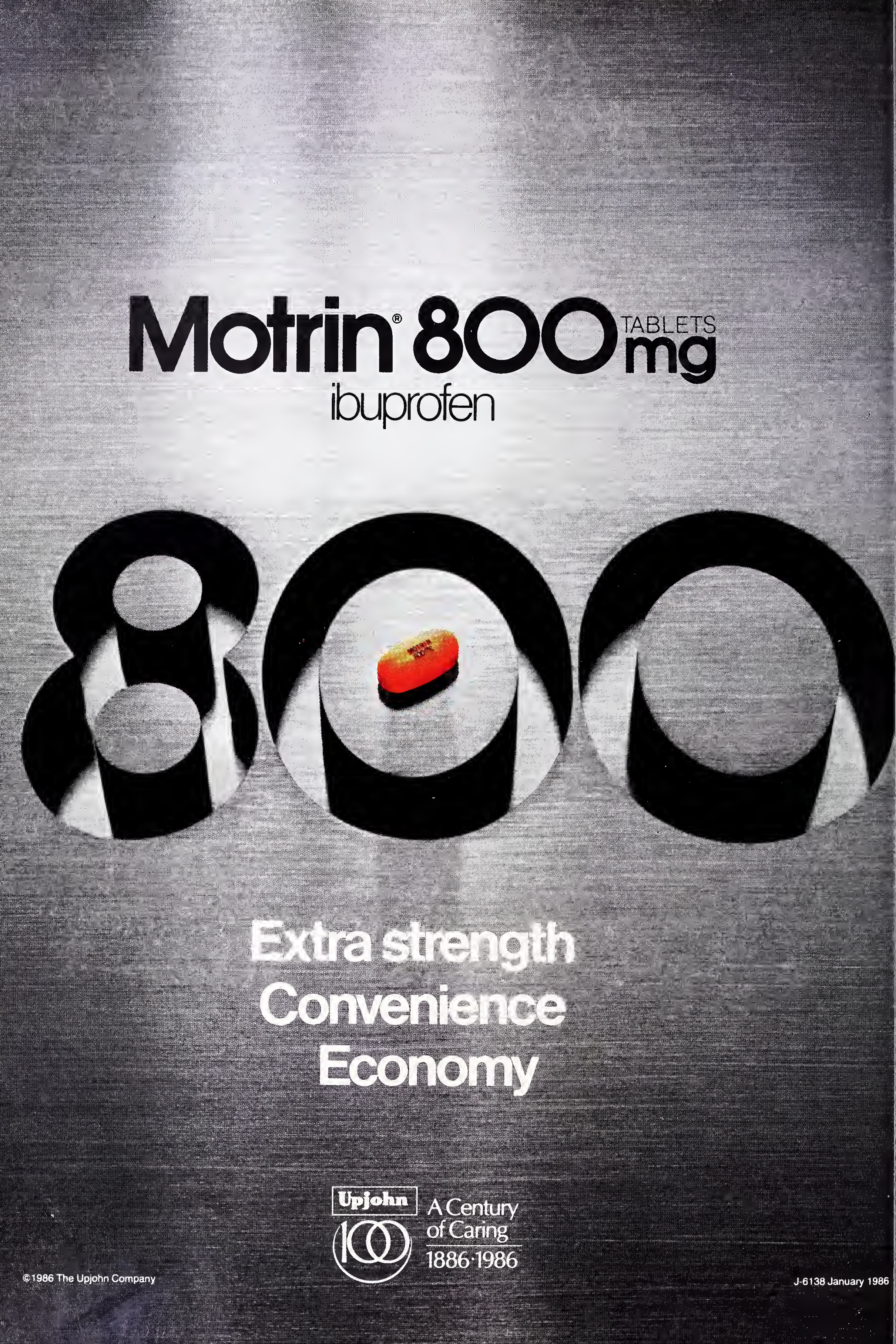
CONCLUSION

In the mid and late 1970s, several studies revealed that one of the healthiest groups of people in the world are the Hunzas who live in the remote mountain villages of northern India. The Hunzas live longer than persons in any other country; they remain active, even working in their fields sometimes into their 80s and 90s. They have no doctors, nurses, hospitals, or drugs. Their peculiar secret is their living habits and this is what I have been discussing. A general study on health done in California in the early 1980s brought forth a suggested model to promote good health and I shall just mention these principles: do not smoke; drink alcohol not at all or only moderately; eat breakfast; do not eat between meals; exercise regularly; avoid becoming overweight; and get at least seven hours of sleep daily. We might call that the Hunza protocol.

As President, I should like to reiterate that I still am deeply concerned about the intrusion of government into the practice of medicine. I have been in practice for 35 years and, therefore, had some experience with practice before the big surge of government intervention some 20 years ago. I do not find nor appreciate that I have treated anyone differently since Medicare, Medicaid, PSRO, HSA, or DRG have come along. Furthermore, I do not believe that any of these programs has improved the practice of medicine nor the care that a patient has received. What I do see is that these programs have squeezed the taxpayer unmercifully, helped to almost bankrupt the Social Security program, and have contributed to an unwieldy federal deficit. The last thing we need at this time is another government program.

My deep personal feeling is that the Medical Society of New Jersey and its membership should be alerting the people of our fine state that we stand ready to care for them in the best possible personal manner (care with caring) regardless of whether there are government programs or not.

In closing, I should like once again to thank you for the opportunity to address you, and furthermore to thank you for the distinguished honor you grant me as your President for the coming year. I pledge to you that I will do my best for the Medical Society of New Jersey, the physicians of New Jersey, and, in particular, for the patients whom we serve.



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J-6138 January 1986

FAREWELL ADDRESS: THE CHANGING SCENE

RALPH J. FIORETTI, M.D., ROCHELLE PARK

Ralph J. Fioretti, M.D., presented this Address at the Annual Meeting of the Medical Society of New Jersey at Americana Host Farm Resort, Lancaster, Pennsylvania, on May 2, 1986.

Was it only a year ago that we left here, determined and inspired, saying to each other—"The days of complacency are over"? They are—and we knew it! On that day, I felt it a great privilege, as President, to represent this Medical Society. But, I quickly realized that it is a responsibility not to be taken lightly, or merely ceremoniously. And, no matter how prepared one hopes to be, there never would be enough time or enough effort for one individual to make the difference he hopes he might. Most of all, he soon learns that nothing ever could be accomplished without the tremendous efforts and sacrifices so many of you have made, not only during the past year, but have been making for our Society for a long time.

During the past year, the officers, councils, committees, and Board of Trustees have devoted a great deal of time to the various aspects of medical practice affecting our members. The results of these deliberations are summarized for you in the Annual Reports, but I would like to share some of these now, very briefly:

Under the capable leadership of Dr. Ralph Kristeller, a major effort was made to bring the message of the rationing of care to senior citizens. We have made an impact. As a result, there have been many hearings by the legislators on the quality of care to senior citizens. The Task Force got the message across to the communities. Some counties have done a great deal; others have done very little. More needs to be done.

As a result of our position on PRO criteria and quality of care, the PRO has written a memorandum to hospitals to stop pressuring and coercing physicians into early and inappropriate discharges. Yet, this same PRO was the originator of this pressure! The Criteria Committee, under the leadership of Dr. Christopher Reilly, has kept us informed. They have challenged some of the PRO methods, pointing out many of the inadequacies of the PRO criteria.

In the area of political action, Dr. William Ryan has done an outstanding job. Our political presence has been expanded significantly. We have played a greater role in the election of state legislators. We have learned that money talks—but early money shouts!

Now that the climate at the Health Department has changed, we look forward to working with Dr. Molly Coye and feel the Medical Society will have a greater input into the system.

For Tort Reform, we have the 14 points as listed by the Commissioner of Insurance.

Changes will occur, not because physicians need relief from the high cost of malpractice insurance, but because liability insurance now affects all segments of the economy.

It should not be doctors against attorneys, it should be people for reform, and we are with the people.

The California case which put a cap on awards for pain and suffering has set the tone for the future.

The federal government is encouraging the states to change tort laws.

We have introduced several bills in the legislature to make these changes.

Health Care Delivery. As you all know, the power has shifted from the doctors and hospitals to the purchasers of health care. Of these, corporate America and the federal government are the biggest. They both are in serious economic trouble. Therefore, they look to buy the cheapest health care package at wholesale prices. Hence, the proliferation of HMOs and PPOs. We cannot sit idly by and wring our hands in despair. We must do as business does. The providers of health care, the physicians and hospitals, must immediately pursue all efforts to become involved in joint ventures—home care, laboratories, cardiac rehab, and surgi-centers.

This window of opportunity for the hospital and physician only will last for a short period. If it is not undertaken together, it will be done by business which is waiting to fill the vacuum. For the physician, in particular, this opportunity will be short-lived, due to the oversupply of doctors almost everywhere. As this supply of physicians grows, government and hospitals will gain the upper hand in any negotiations.

HMO and Joint Ventures. We have held three meetings, one in northern New Jersey, another in Lawrenceville, and a third in Cherry Hill, to enlighten the membership on the pros and cons of the various options under HMOs and the legal implications to the physicians. It is projected that 30 percent of the population will be enrolled in an HMO by 1990. As a result of these seminars held by Dr. Bernard Robins and his committee, there has become an awareness that perhaps the Medical Society of New Jersey should look into a plan for implementation of our own HMO. This problem will be brought to this House of Delegates this weekend for your deliberations.

Certificate of Need. We feel we will prevail in preventing this legislation from being extended to physicians. The certificate of need process soon will be abandoned by Congress which no longer will support this concept, since regulations concerning capital, graduate medical education, and all other components will take over and there no longer will be a need for the regulated planning system, because hospitals will have limited capacity to engage in capital projects.

There is better understanding and communication between the Medical Society and the Board of Medical Examiners. This is due in great part to the leadership of Dr. Edward W. Luka.

Finally, I would like to close with a word of hope and a deep sense of appreciation to all of you. Our Medical Society is strong and, because of you, we are getting stronger. Every one of you cares. You are responding to the challenges we face.

The most pressing issue of our time is to give quality care at affordable prices. Four million patients see doctors every day. These are four million opportunities for us to show the public how much we care. The average doctor gives $18,000 worth of free care every year, some much more. Stay with the Art and Form of medicine. Continue to give our patients more good care, even though it is in a business atmosphere. This is no time for fear. Ours is a passion for excellence. Our critics tie our hands and then blame us for not resolving the problems. We must insert reason where chaos is beginning to prevail.

We must join ranks and work together. Physician unity is absolutely essential if we are to succeed in this crisis. We must continue to make a greater effort to expand our membership.

I would like to thank all the members of the Society throughout the state who have helped me. Your counties welcomed me everywhere and offered their assistance as well as their hospitality. I owe my own Bergen County a great deal. They and George Willis always have supported me in every way possible.

As to what I had wished to accomplish and what I did, I am uncomfortably aware of the hiatus between the goal and the performance. But I can accept this, because I know how hard we have all worked.

One of the biggest rewards of this office is the opportunity of serving with such professional people—our Executive Director, Vincent Maressa, and his competent staff, expecially Diana Gore. I would have been helpless without them. And, of course, the gracious Eileen Pfeiffer. She and Dr. Frank Romano had to overcome many problems this year in order that we might be accommodated for this convention. I thank each and every one of them who, for the most part, are unrecognized and unrewarded for their special dedication to us.

But, most of all, I will always have a profound sense of gratitude to the officers, Board of Trustees, councils, committees and, especially, the Fellows.

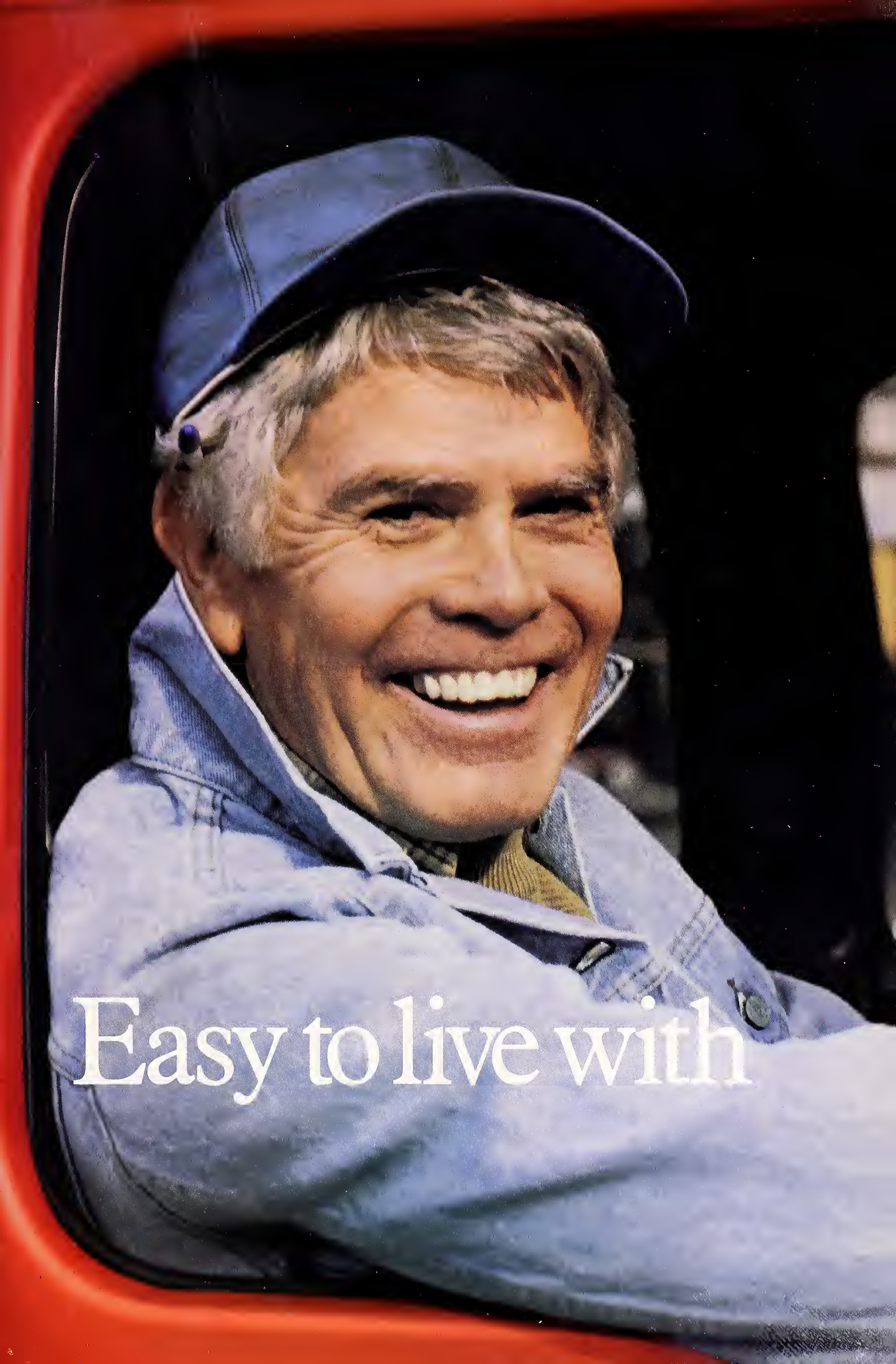
Because of all these wonderful people, the Medical Society of New Jersey is in good hands.

Not far from here is a sign that reads "The Road to Success is always under construction."

And, so I extend my best wishes to the Incoming President, Dr. Edward Schauer, for a successful and rewarding year.

Let us ask the good Lord to help us to be true to our profession, to those who come to us for help, to ourselves, and to each other.

My heartfelt thanks to everyone.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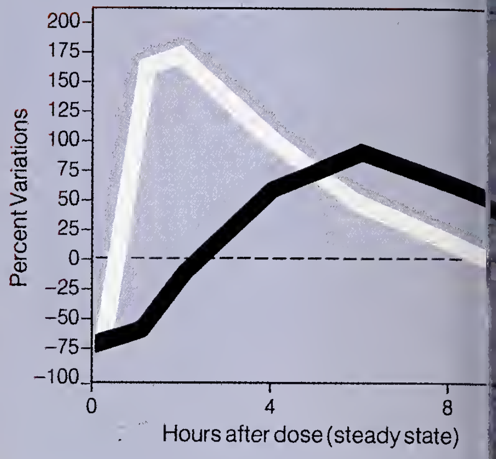

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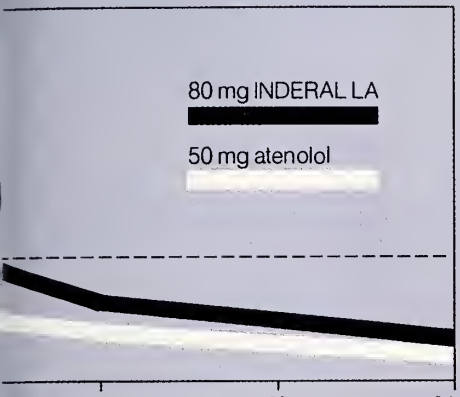

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CONTRAINDICATIONS

Propranolol hydrochloride (INDERAL® LA): Propranolol is contraindicated in: 1) cardiogenic shock; 2) sinus bradycardia and greater than first degree block; 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with propranolol.

Hydrochlorothiazide: Hydrochlorothiazide is contraindicated in patients with anuria or hypersensitivity to this or other sulfonamide-derived drugs.

WARNINGS

Propranolol hydrochloride (INDERAL® LA): CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated, and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or propranolol should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction following *abrupt* discontinuance of propranolol therapy. Therefore, when discontinuance of propranolol is planned the dosage should be gradually reduced and the patient carefully monitored. In addition, when propranolol is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If propranolol therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute propranolol therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD, IN GENERAL, NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution, since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

DIABETES AND HYPOGLYCEMIA: Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin. Hypoglycemic attacks may be accompanied by a precipitous elevation of blood pressure.

Hydrochlorothiazide: Thiazides should be used with caution in severe renal disease. In patients with renal disease, thiazides may precipitate azotemia. In patients with impaired renal function, cumulative effects of the drug may develop.

Thiazides should also be used with caution in patients with impaired hepatic function or progressive liver disease, since minor alterations of fluid and electrolyte balance may precipitate hepatic coma.

Thiazides may add to or potentiate the action of other antihypertensive drugs. Potentiation occurs with ganglionic or peripheral adrenergic-blocking drugs.

Sensitivity reactions may occur in patients with a history of allergy or bronchial asthma.

The possibility of exacerbation or activation of systemic lupus erythematosus has been reported.

PRECAUTIONS

Propranolol hydrochloride (INDERAL® LA): GENERAL: Propranolol should be used with caution in patients with impaired hepatic or renal function. Propranolol is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that propranolol may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs, such as reserpine should be closely observed if propranolol is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity, which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies, in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. Propranolol has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximal recommended human dose. There are no adequate and well-controlled studies in pregnant women. Propranolol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

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NURSING MOTHERS: Propranolol is excreted in human milk. Caution should be exercised propranolol is administered to a nursing mother.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

Hydrochlorothiazide: GENERAL: Periodic determination of serum electrolytes to c possible electrolyte imbalance should be performed at appropriate intervals.

All patients receiving thiazide therapy should be observed for clinical signs of fluid or elect imbalance, namely: Hyponatremia, hypochloremic alkalosis, and hypokalemia. Serum and electrolyte determinations are particularly important when the patient is vomiting excessiv receiving parenteral fluids. Medication such as digitalis may also influence serum electro Warning signs irrespective of cause are: Dryness of mouth, thirst, weakness, lethargy, drowsi restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardia gastrointestinal disturbances such as nausea and vomiting.

Hypokalemia may develop, especially with brisk diuresis, when severe cirrhosis is prese during concomitant use of corticosteroids or ACTH.

Interference with adequate oral electrolyte intake will also contribute to hypokalemia. H kalemia can sensitize or exaggerate the response of the heart to the toxic effect of dig (eg, increased ventricular irritability). Hypokalemia may be avoided or treated by use of potas supplements, such as foods with a high potassium content.

Any chloride deficit is generally mild and usually does not require specific treatment, e under extraordinary circumstances (as in liver or renal disease). Dilutional hyponatremia may in edematous patients in hot weather; appropriate therapy is water restriction, rather than adn tration of salt, except in rare instances when the hyponatremia is life-threatening. In actua depletion, appropriate replacement is the therapy of choice.

Hyperuricemia may occur or frank gout may be precipitated in certain patients receiving thi therapy.

Insulin requirements in diabetic patients may be increased, decreased, or unchanged. Diab mellitus which has been latent may become manifest during thiazide administration.

If progressive renal impairment becomes evident, consider withholding or discontinuing diu therapy.

Thiazides may decrease serum PBI levels without signs of thyroid disturbance.

Calcium excretion is decreased by thiazides. Pathologic changes in the parathyroid gland hypercalcemia and hypophosphatemia have been observed in a few patients on prolo thiazide therapy. The common complications of hyperparathyroidism, such as renal lithiasis, resorption, and peptic ulceration, have not been seen. Thiazides should be discontinued b carrying out tests for parathyroid function.

DRUG INTERACTIONS: Thiazide drugs may increase the responsiveness to tubocurarine.

The antihypertensive effects of thiazides may be enhanced in the postsympathectomy pa Thiazides may decrease arterial responsiveness to norepinephrine. This diminution is not suffi to preclude effectiveness of the pressor agent for therapeutic use.

PREGNANCY: Pregnancy Category C. Thiazides cross the placental barrier and appear in blood. The use of thiazides in pregnancy requires that the anticipated benefit be weighed ag possible hazards to the fetus. These hazards include fetal or neonatal jaundice, thrombocytop and possibly other adverse reactions which have occurred in the adult.

NURSING MOTHERS: Thiazides appear in human milk. If use of the drug is deemed esse the patient should stop nursing.

PEDIATRIC USE: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Propranolol hydrochloride (INDERAL® LA): Most adverse effects have been mild transient and have rarely required the withdrawal of therapy.

Cardiovascular: Bradycardia; congestive heart failure; intensification of AV block; hypoten paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Rayr type.

Central Nervous System: Lightheadedness; mental depression manifested by inson lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; vi disturbances; hallucinations; an acute reversible syndrome characterized by disorientatio time and place; short-term memory loss; emotional lability; slightly clouded sensorium; decreased performance on neuropsychometrics.

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, co pation; mesenteric arterial thrombosis; ischemic colitis.

Allergic: Pharyngitis and agranulocytosis; erythematous rash; fever combined with aching sore throat; laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis; nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reporte

Miscellaneous: Alopecia; LE-like reactions; psoriasiform rashes; dry eyes; male impotence; Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the serous membranes, and conjunctivae reported for a beta blocker (practolol) have not b associated with propranolol.

Hydrochlorothiazide:

Gastrointestinal: Anorexia; gastric irritation, nausea, vomiting, cramping, diarrhea, constipa jaundice (intrahepatic cholestatic jaundice); pancreatitis; sialadenitis.

Central Nervous System: Dizziness, vertigo; paresthesias; headache; xanthopsia.

Hematologic: Leukopenia; agranulocytosis; thrombocytopenia; aplastic anemia.

Cardiovascular: Orthostatic hypotension (may be aggravated by alcohol, barbiturates narcotics).

Hypersensitivity: Purpura; photosensitivity; rash; urticaria; necrotizing angiitis (vascu cutaneous vasculitis); fever; respiratory distress, including pneumonitis; anaphylactic reactior

Other: Hyperglycemia; glycosuria; hyperuricemia; muscle spasm; weakness; restlessn transient blurred vision.

Whenever adverse reactions are moderate or severe, thiazide dosage should be reduce therapy withdrawn.

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NURSING HOME REGULATION IN NEW JERSEY: CASE ANALYSIS OF PUBLIC POLICYMAKING

NANCY A. BUBRICK, M.P.A., NEWPORT, RI*
JOHN A. WORTHLEY, D.P.A., SOUTH ORANGE*

The state of New Jersey presently is considering legislation to limit the use of private pay contracts by the nursing home industry.† This article concludes that enactment of such legislation is likely to produce serious unintended consequences.

The state of New Jersey is considering legislation† which would prohibit the use of private pay contracts by the nursing home industry. This article develops guidelines for long-term care public policies by analyzing Bill ACS-1829 as a case study. The focus is on the impact of this bill, intended as well as unintended. To determine the likely impact, data were collected from the nursing home industry as to what types of actions, if any, would be taken in response to enactment of this legislation.

Nursing home care for the elderly is an essential and costly component of long-term care. Persons unable to pay for such care eventually resort to the use of public funds for payment.

Despite the promises of Medicaid, many persons are finding that, without a good deal of money, there are no beds to be found. Since 1979, the monthly list of persons awaiting nursing home placement in New Jersey has averaged 2,750 people.[1]

Nursing homes typically deny admission to a Medicaid patient unless the family agrees to forego the patient's right to Medicaid reimbursement and pay the higher private pay rate for a specified period of time, sometimes for as long as four years; ACS-1829 is designed to address this situation.

BACKGROUND OF ACS-1829

The proposed policy is intended to protect a prospective Medicaid patient and/or the family who cannot afford to pay for nursing home care.[2]

The major highlights of Bill ACS-1829 are as follows:

- Administrators and operators of nursing homes are forbidden from requiring incoming Medicaid-eligible patients to sign private pay contracts if the facility does not have 45 percent of its population as Medicaid patients. The second year, the rate would increase to 50 percent; the third year to 55 percent.
- Penalties will be imposed upon the administrator or operator found guilty of violation. The violator would be guilty of a third-degree crime, and would be liable to the persons forced to sign the contract for the amount, plus interest, paid out in compliance with the contract. The violator also would be liable for a penalty of $5,000 for each claim.

Concern over ACS-1829 prompted this study.

METHODOLOGY

In February 1985, following the New Jersey State Assembly passage of ACS-1829, a one-page questionnaire was mailed to the administrators of 194

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†Bill was signed into law in August 1985.

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TABLE 1

Responses of Nursing Home Administrators

	Yes		No	
	Percent	**Number**	**Percent**	**Number**
1. Are you in favor of this bill?	8.3	6	91.7	66
2. Are Medicaid reimbursement rates lower than private pay rates?	100.0	68	0	0
3. Are Medicaid reimbursement rates adequate to cover costs?	5.9	4	94.1	64
4. Are Medicaid reimbursement rates adequate to maintain a profit?	1.5	1	98.5	67
5. Do you feel that private pay contracts in New Jersey resulted from:				
a) an inadequate supply of beds?	22.7	10	77.3	34
b) inadequate Medicaid reimbursement rates?	95.5	63	4.5	3
6. Do private pay patients supplement losses from services rendered to Medicaid patients?	92.6	63	7.4	5
7. If this bill is passed, will you:				
a) increase private pay rates?	85.5	47	14.5	8
b) withdraw from Medicaid:	35.1	13	51.4	19
noted "are considering withdrawal"	13.5	5		
8. Would passage of this bill promote equal access for all patients regardless of payment source?	9.1	6	90.9	60

proprietary nursing homes in New Jersey. The questionnaire was accompanied by a cover letter describing the Medicaid quota requirements of the bill, and a return self-addressed, stamped envelope. In order to ensure confidentiality, the only identifiable information solicited was a count of the number of patients in each facility, categorized by method of payment.

A questionnaire, prepared in the same manner, was mailed to the chief executive officer (CEO) of 20 major investor-owned nursing homes systems in the United States. The only identifiable information solicited in this questionnaire was whether they presently owned or managed any nursing homes in New Jersey.

LIMITATIONS

Less than one week after the questionnaires were mailed, the New Jersey Association of Health Care Facilities (NJAHCF), the industry's trade association, issued a written memorandum to all member facilities advising them not to complete the questionnaire.

The president of NJAHCF was contacted by telephone by the authors. During the discussion, the president refused to rescind his memorandum and indicated that the responses to one question on this survey could expose the industry to anti-trust violations. The objection was to a question which sought information about the specific actions that would be taken in response to enactment of this legislation.

By mid-March, a 37 percent return rate had been achieved. Of the total 72 surveys returned, 37 (51.4 percent) had responded to the objectionable question.

A followup letter mailed to the chief executive officers of nursing home systems produced a 25 percent return rate by April.

FINDINGS

Table 1 represents data obtained from the replies of 72 proprietary nursing home administrators. The questionnaire contained a final question, not displayed in Table 1 which sought the number of patients in each facility, categorized by method of payment.

Table 2 represents the responses of five chief executive officers of major investor nursing home systems in the United States.

The first four questions on both surveys were designed to verify the accuracy of information acquired from the literature. The results were not unexpected. The majority of responding administrators and chief executive officers are opposed to ACS-1829 and indicate that Medicaid reimbursement rates are inadequate to cover costs.

The majority of respondents to both surveys cite inadequate Medicaid reimbursement rates as a cause of private pay contracts. The responding chief executive officers of nursing home systems who presently are not involved with New Jersey facilities indicate that private pay contracts were a result of both an inadequate bed supply and inadequate Medicaid reimbursement rates.

The majority of respondents to both surveys indicated that private patients supplement losses from services rendered to Medicaid patients. The industry maintains that an appropriate mix of patients is necessary to maintain a profit and provide quality care.

Each survey contained a question which sought specific information regarding action that would be taken in response to the enactment of ACS-1829. Nursing home administrators did not appear reluctant to answer this question. The surveys which were completed prior to the memo distributed by the New Jersey Association of Health Care Facilities contained replies to this question. Typically, the surveys which were returned later did not contain responses.

Over 85 percent of the responding administrators

TABLE 2

Responses of Chief Executive Officers of Investor-Owned Nursing Home Systems

	Yes		No	
	Percent	Number	Percent	Number
1. Are you in favor of this bill?	0	0	100	5
2. Are New Jersey Medicaid reimbursement rates lower than private pay rates?	100	5	0	0
3. Are New Jersey Medicaid reimbursement rates adequate to cover costs?	0	0	100	5
4. Are New Jersey Medicaid reimbursement rates adequate to maintain a profit?	0	0	100	5
5. Do you feel that private pay contracts in New Jersey resulted from:				
a) an inadequate supply of beds?	60	3	40	2
b) inadequate Medicaid reimbursement rates?	100	5	0	0
6. Do private pay patients supplement losses from services rendered to Medicaid patients?	100	5	0	0
7. Do you own or manage nursing homes in New Jersey?	60	3	40	2
8. If yes, if this bill is passed will you:				
a) increase private pay rates?	33.3	1	66.7	2
b) withdraw from Medicaid program?	0	0	100	2
9. Would passage of this bill deter you from future involvement with New Jersey nursing homes?	100	5	0	0
10. Would passage of this bill promote equal access for all patients regardless of payment source?	20	1	80	4

and 33.3 percent of the responding chief executive officers indicated that they plan to increase private pay rates. If such action is taken, some patients will have difficulty paying for nursing home care with their own resources.

It is possible that this action will result in increased length of hospital stays and an enlarged nursing home population which must be supported by Medicaid funds.

Many patients seek temporary nursing home placement prior to returning home from the hospital. If unable to afford this care, some patients will be forced to remain in the hospital longer. Longer hospital stays may have negative financial impacts upon New Jersey hospitals and are in conflict with the objectives of DRG methods of reimbursement.

Private pay patients in nursing homes will exhaust their resources earlier, thus qualifying for Medicaid benefits. The rate of conversion of private paying patients to Medicaid status will be accelerated.

Over 35 percent of the administrators who responded to question 7 indicated that they plan to withdraw from the Medicaid program. An additional 13.5 percent noted that they are considering withdrawal. None of the CEOs presently involved with New Jersey facilities plan to withdraw from Medicaid.

Two possible explanations exist for these conflicting results. First, although a high percentage of administrators indicated that they plan to withdraw, the likelihood of this actually coming to pass is unknown. It is suggested here that their response may have been designed to prevent passage of ACS-1829. Secondly, it is possible that the responding CEOs were aware of the implications of answering affirmatively to this question, i.e. antitrust violations.

An immediate impact will ensue if providers refuse to participate in the Medicaid program. At a minimum, there will be 385 persons forced to leave these nursing homes unless there is some individual willing and able to take over the private costs of remaining in the nursing home. If we account for the facilities that are considering withdrawal, the total number of patients needing relocation rises to 599. It is significant to note that these figures are an underestimation since five of the respondents did not provide data on the number of patients presently in their facilities. Projected for the entire Medicaid population of 8,500 patients in proprietary nursing homes in New Jersey, 2,983 people will be forced out of nursing homes if they are unable to assume the private costs of such care.

One of several scenarios will occur in the case of a Medicaid patient facing eviction from a nursing home because of withdrawal from the program. For some patients, their families may be capable of assuming total responsibility for the care of these patients. Some family members will pay for continued nursing home care, or will provide the necessary care enabling the patient to re-enter the community.

Patients without the support of family, or without family members to rely upon, may be able to draw upon existing resources and return to the community. This does not appear likely since the individual is without financial resources and without housing.

Far more probable are the negative impacts which may occur. It is not unrealistic that there will be an increase in the number of unnecessary hospitalizations of this type of patient, or the abandonment by family members unable to assume the responsibility of becoming primary caregivers.

The forced relocation of Medicaid nursing home patients would have a tremendous emotional impact. Elderly individuals will be forced to sever relationships which have been formed with staff and fellow residents of the nursing home. Familiar surroundings and daily routines will be disrupted. It is unlikely that relocation efforts will be less than traumatic for the elderly person who has exhausted all resources and has adjusted to life in a nursing home.

There also will be an impact upon the Medicaid-eligible person seeking admission to a nursing home. With fewer facilities willing to accept Medicaid patients, the problems already associated with the access issue will be exacerbated. Hospital backup days will increase and increasing numbers of patients at home will continue to deteriorate.

Over 90 percent of the responding nursing home administrators and 80 percent of the CEOs of home nursing systems do not believe that ACS-1829 will promote equal access for nursing home patients, regardless of payment source. The private paying patient may have difficulty securing a bed, producing a form of reverse discrimination.

The findings of the survey of investor-owned nursing home systems indicate impacts that will conflict with the recommendations of the New Jersey Nursing Home Task Force.

The Task Force has substantiated the perception that New Jersey suffers from a shortage of long-term beds. Population trends indicate that this shortage is likely to worsen. Future construction of nursing homes is one alternative suggested by the Task Force to eliminate this shortage. The survey findings suggest that 100 percent of the CEO respondents will be deterred from future involvement with New Jersey nursing homes if ACS-1829 is enacted.

CONCLUSION

Further systematic research is needed in several areas concerning the unintended consequences of ACS-1829. As a first step, the state is urged to pursue further studies which will test the validity and probability of the findings of this case study. If the findings of this study are validated by further research, it then is recommended that the state consider available mechanisms to deal with the retaliatory actions, such as the enforcement of federal anti-trust and civil rights laws.

Should continued research show that serious unintended consequences may occur, and that available options to deal with the problems are inadequate, consideration must be given to alternative methods of eliminating the access problem. Experience tells us that it is far more prudent to prevent bad policy than to undo policy failures.

Nursing home policy and the nursing home market are complex. As in several other arenas, government relies upon private business for the provision of necessary goods and services. While nursing homes are dependent on government for most of their income, the governments, in turn, are dependent on many private nursing homes for the provision of services. A crucial element of nursing home politics must be understood by those who seek to affect policy, i.e. that proprietary nursing homes do not stay in business to lose money.

Policymakers must be cognizant of the fact that any government action which will affect the ability of private nursing homes to maintain a profit will be met with political pressure and retaliatory action. Unless the government chooses to provide all services directly, it must contend with the interests of the proprietary sector.

There exists an abundance of literature which addresses the long-term care policy problems in this country.[4] A reiteration of the recommendations, at the very least, is redundant.

It is recommended, however, that further research into these issues must precede enactment of legislation with possible serious unintended consequences. Specifically, the issues which warrant consideration are: the lack of coordinated, accessible home care; Medicaid's bias towards institutionalization; and Medicaid reimbursement system.

The first two issues relate to the inappropriate utilization of nursing home care. A 1976 study of New Jersey Medicaid nursing home patients concluded that more than 1,700 persons could be cared for at lower levels of care, thus opening up beds for others awaiting placement.[5]

The third issue, Medicaid reimbursement systems, is controversial, mainly due to tightening of the Medicaid budget. It must be viewed, however, as an effective means for government to encourage, rather than demand, desired behavior from the nursing home industry. It is recommended that consideration be given to a system of reimbursement differentials which would provide incentives for nursing homes to admit a higher percentage of Medicaid patients.

The information presented in this study clearly indicates the need for further research in order to achieve equity for all persons needing nursing home care.

Ideally, long-term care policy should guarantee the right to appropriate services, in or out of an institution. Future policy should be based upon the assumption that not everyone needs or desires nursing home care. Policymakers are urged to consider the expansion of noninstitutional care.

Demographic trends and budgetary limitations will most likely intensify the problems which exist today. It therefore is essential that policy reforms be formulated, implemented, and monitored. To ensure that policy reforms are prudent and effective, further research is recommended to assess the impact of such changes on the providers and the beneficiaries of long-term care.

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RENAL ONCOCYTOMA

JOEL W. ROSENBERG, M.D., CLIFTON*

Renal oncocytoma is a rare benign kidney tumor. It must be distinguished from renal cancer as the prognosis differs radically. Three cases are reported from one suburban community.

Renal oncocytoma is a rare benign kidney tumor. There are less than 200 cases accumulated in the world literature. The pathologic features are unique and readily distinguishable from malignant renal tumors. The radiologic features are not as distinctive and most cases are treated as malignant tumors until the pathologist finds otherwise.

This report presents three cases from a small suburban community and is remarkable in that the author, who treated two of the patients, was himself the third case.

CASE REPORT 1

A 67-year-old man presented with gross hematuria and obstructive voiding symptoms. An intravenous pyelogram suggested a small right renal lesion. Cystoscopy disclosed an obstructing prostate which was resected. One chip was positive for adenocarcinoma; the others were all benign (15 gm).

A CT scan and arteriogram confirmed a small hypovascular solid lesion of the right kidney (Figures 1A and 1B). Transabdominal nephrectomy was performed and the subsequent pathology was renal oncocytoma, confirmed by the Armed Forces Institute of Pathology (Figure 2).

CASE REPORT 2

A 67-year-old man had a long history of obstructive urinary symptoms. An intravenous pyelogram prior to suprapubic prostatectomy for benign prostatic hypertrophy (BPH) revealed a "cyst" of the right kidney. However, a CT scan (Figure 3) revealed the lesion to be solid and a right transabdominal nephrectomy was performed. Pathology was oncocytoma (Figure 4).

CASE REPORT 3

A 37-year-old urologist was found to have a palpable right upper quadrant mass during routine annual physical examination. An intravenous pyelogram and CT scan revealed a large right renal tumor (Figures 5A and 5B). Preoperative urinalysis was normal. The hemoglobin was 10 gm and no cause for anemia was demonstrated.

A right radical thoracoabdominal nephrectomy was performed and the pathology was renal oncocytoma, confirmed by electron microscopy. Postoperatively, the hemoglobin returned to normal.

DISCUSSION

Renal oncocytomas have been described as early as 1962,[1] but the recent spate of reports has been spurred by the report of Klein and Valensi.[2]

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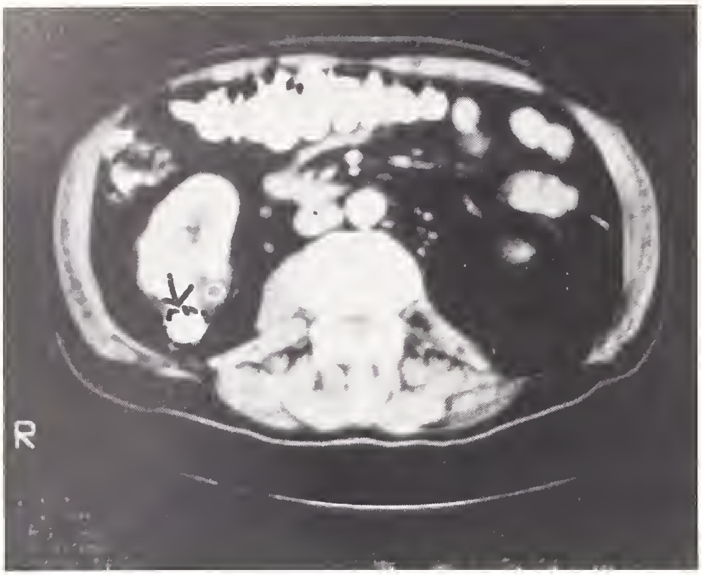

Figure 1A—CT scan with solid lesion of kidney adjacent to lucent cyst of kidney.

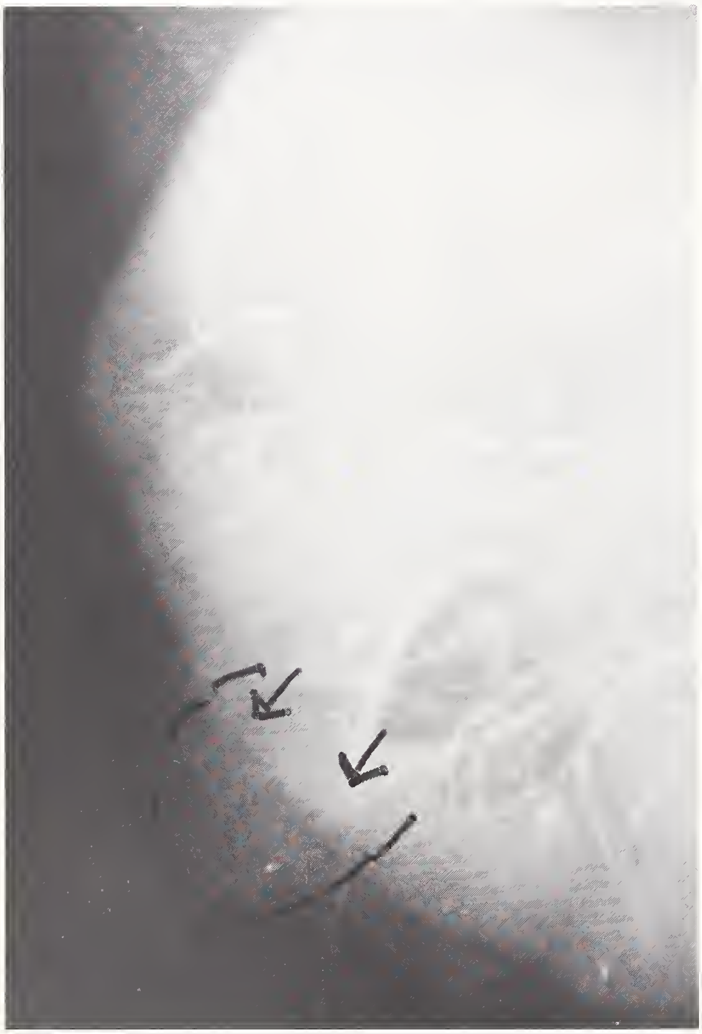

Figure 1B—Renal arteriogram revealing solid lesion of right kidney.

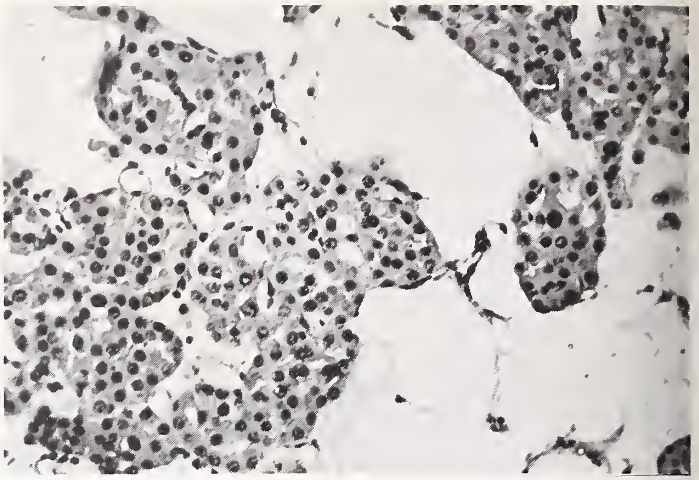

Figure 2—Typical plump monotonous oncocytes.

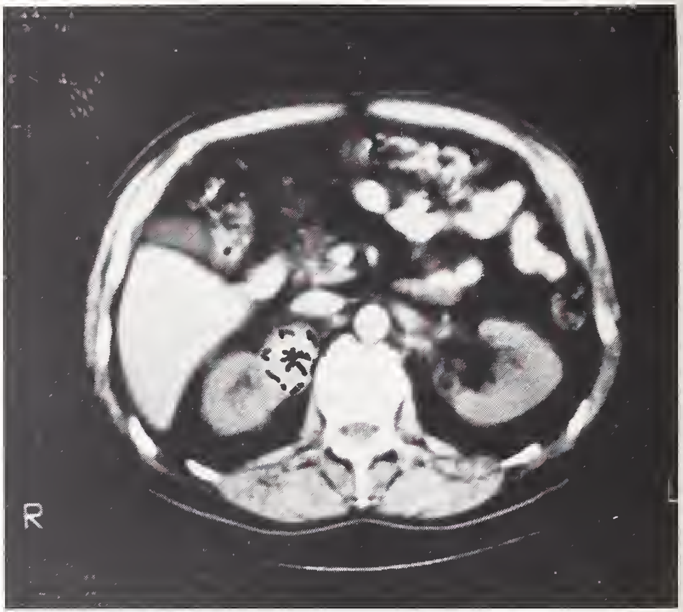

Figure 3—Solid tumor of right kidney on CT scan.

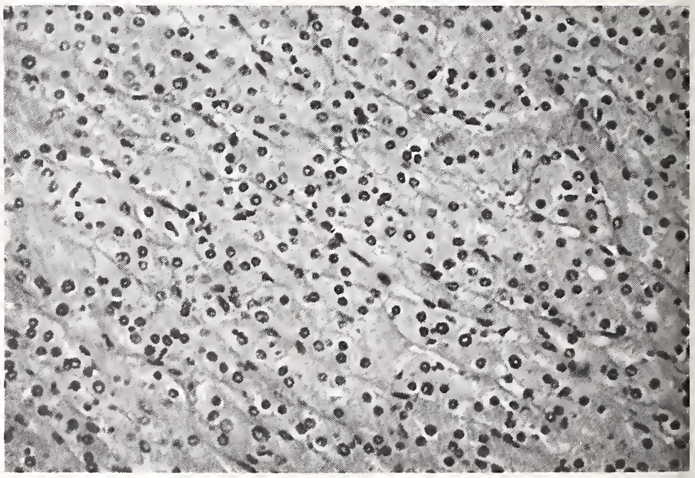

Figure 4—Sheets of oncocytes.

The most common clinical presentation is that of an incidental finding during routine examination or evaluation for other disease processes. It generally is a symptomless disease and hematuria is not a prominent feature. Anemia (case 3) has not been described previously in conjunction with this tumor.

Radiologic features of distinctive ("spoke wheel") vasculature are described,[3] but the angiographic findings are nonspecific, or ill-defined at best.

The gross and microscopic pathology as well as electron microscopic findings are all distinctive. Grossly, the tumor is a mahogany color. The histologic picture is that of plump granular eosinophilic "oncocytes." Under the electron microscope, the most striking feature is the abundance of mitochondria.

Treatment consists of surgical extirpation. Ideally, a renal sparing operation (partial nephrectomy) could be recommended if the diagnosis were secure preoperatively but this is seldom the case. The incidence of synchronous bilateral lesions is high (10 percent) and

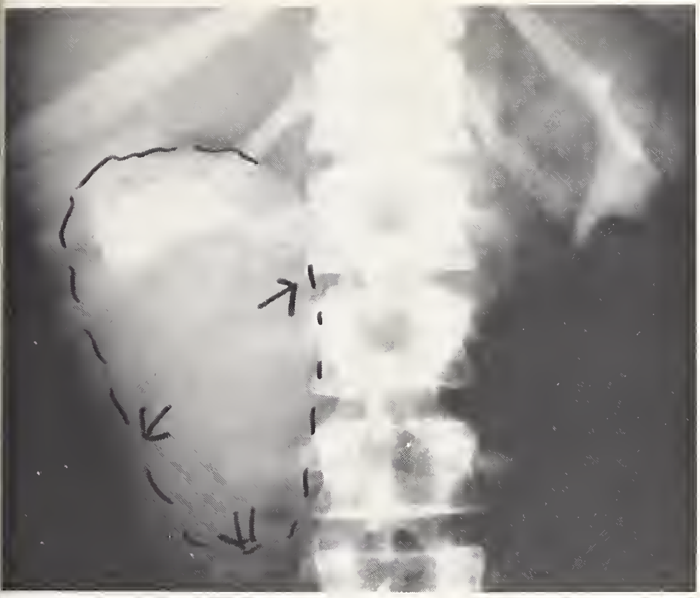

Figure 5A—Huge right renal mass.

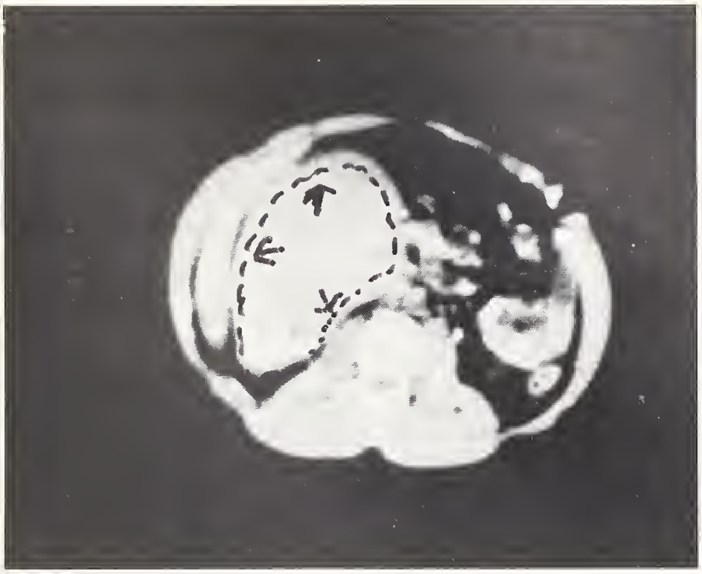

Figure 5B—Same lesion on CT scan filling entire right half of abdomen.

in these cases, partial nephrectomy has been used to spare renal function when the diagnosis was known or presumed.[3,4]

No case to date has demonstrated local invasion or distant metastases and clinically there is no reason to doubt the benignity of the lesion. Recurrence has been reported in the contralateral kidney. No patient has died of the disease.

Several authors have indicated the possibilities of clusters[4,5] and increasing incidence of this tumor. This may be due to increased awareness of the diagnosis rather than to a true increase in incidence. However, this report with three patients from a small suburban community, despite true statistical significance, must raise the specter of "tumor cluster" and cause one to pause in regard to the etiology of this rare disease.

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STATE OF THE ART: EARLY INTERVENTION AND PHYSICIAN REFERRAL

ANTHONY J. LATESS, M.ED., AND PATRICIA MUNDAY HILL, ED.D., PISCATAWAY*

Landmark legislation in New Jersey mandates the provision of intervention programs and services to developmentally disabled children from birth to five years. This article presents information that will help physicians understand the nature, value, and benefits of these services.

Landmark legislation in New Jersey (Chapter 415 of the laws of 1981), which became effective July 1, 1983, has caused a rapid expansion in services to handicapped infants and their families. Chapter 415 mandates the provision of programs to developmentally disabled children from birth to five years of age and establishes two separate service delivery systems for these children. Local public school districts are responsible for providing special education programs and related services for three- to five-year-old handicapped children. Children below this age, from birth to three years of age, receive services through public and private agencies cooperating under contract with the State Departments of Education, Health, and Human Services.

HISTORY OF EARLY INTERVENTION

New Jersey is one of the first states to provide a network of free early intervention programs for handicapped infants and toddlers. This has come about because of New Jersey's impressive history in providing such services to handicapped infants and their families. Approximately 12 years ago, the New Jersey Departmental Disabilities Council identified gaps in the service delivery system for the birth-to-three developmentally delayed population. At that time, there were a few pilot programs on a national level and a limited number of such programs for handicapped children in New Jersey under the age of five. Through the years, there has been a constant growth and expansion; now, residents of all counties have access to a funded program.

The New Jersey legislature has increased dramatically the amount of money available for services to handicapped infants and toddlers. These services have been phased in with the first state appropriations of slightly under two million dollars in 1983 to 1984. This appropriation has increased yearly with the current year appropriation of ten million dollars for 1985 to 1986. This expanded funding has allowed a yearly increase in the number of infants receiving services from 1,000 in 1983 to 1984 to a projected 3,000 in 1985 to 1986.

BENEFITS OF EARLY INTERVENTION

Significant research has been conducted over the last 50 years that provides increasing documentation regarding the value and benefits of early intervention. Research by Kelly on the effects of a handicapped child on the family indicates that: parents of disabled children have unique needs; parent/infant interaction is crucial to infant development; and parents should understand their child's health and development status.[1]

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Brazelton reports that services to parents in intervention programs help them convert their feelings of grief over the child's handicap into a desire to acquire parenting skills to enhance their child's development.[2] Other children in the family show less disturbance and are better able to adapt and cope.

Efficacy studies of early intervention programs suggest that important program outcomes include the strengthening of mother/child attachment and an increase in the quality of the pair's interaction. These changes in turn are thought to lead to improved child progress. Family involvement, therefore, is a major component of early intervention services. Programs provide opportunities for parents of developmentally delayed children to meet together weekly to share experiences, gain emotional support, and acquire skills in coping adaptively. Through active participation in the child's program, they also learn useful information regarding ways to enhance their comfort in caregiving and managing the behavior of the infant.

The normal newborn is able to attend to and discriminate among stimuli with all of the senses. Increasingly, the normal infant shows progressive development in the motor, emotional, social, language, and cognitive domains. Development in these domains is not discrete, and each area of development is difficult to separate from the others; thus a handicap in one domain may impinge on development in other areas. Benjamin Bloom analyzed several longitudinal studies and concluded that 50 percent of all intelligence is developed by age five and 80 percent by age eight.[3] The impact of experience on brain development is substantial and although the effects of some diseases and conditions cannot be eliminated or reversed, they can be minimized and other compensatory areas of function developed while the child still is very young.

The National Handicapped Children's Early Education Program tracked a group of 9,600 biologically impaired children representing a wide array and degree of handicaps.[4] In addition to earlier intervention, the report cited a significant advantage to more accurate diagnosis of a child's handicaps through longitudinal educational observations.

Skeels and Dye studied the effects of early intervention on two groups of retarded infants aged two years or less in Iowa.[5] Thirteen infants were placed—one to a ward—with adolescent, institutionalized girls. These infants received attention and developmental stimulation from the retarded girls and the ward attendants. Twelve other infants were left in the orphanage and "exposed to a relatively nonstimulating environment over a prolonged period of time." In 1942, 18 months later, those children receiving attention and stimulation gained an average of 28 I.Q. points; those left in the orphanage, unstimulated, dropped an average of 26 I.Q. points.

In a followup study by Skeels, all cases were located after a lapse of 21 years.[6] Those in the experimental group were all self-supporting; 11 were married. Of the contrast group, only 2 were married. The median grade completed by the experimental group was 12. The median grade completed by the contrast group was less than 3. The cost to the state of the contrast group was approximately five times that of the experimental group.

In 1981, Schweinhart and Weikart reported on their 15-year followup study of 123 subjects from age 3 to age 19. In 1962, these children, who were functioning in the mildly mentally retarded range of intelligence, were randomly assigned to an experimental preschool program group or to a control group which received no early childhood program. Children who attended the Perry Preschool Program showed significant gains, including a stronger commitment to schooling, higher scholastic achievement, a 50 percent reduced need for special education services, and less antisocial or delinquent behavior outside of school.

Each of these studies has implications for cost effectiveness. There is clear evidence to support the fact that the cumulative cost in human productivity and community savings is overwhelmingly less when intervention begins early.

LIMITATIONS OF RESEARCH

In considering these data, it must be recognized that there are limitations in conducting research in the area of early intervention including: the lack of reliable measures, difficulty in the comparability of subjects, and the ethically unacceptable organization of experimental groups of handicapped children who receive specialized intervention and control groups of handicapped children who receive little or no intervention services.

RANGE OF SERVICES TO HIGH-RISK/ HANDICAPPED INFANTS IN NEW JERSEY

The current range of services to handicapped and at-risk infants in New Jersey includes: the level III neonatal intensive care units, high-risk registry, county case management units of special child health services, and early intervention programs. Certain graduates of level III neonatal intensive care units are followed up on a regular basis for the assessment of growth, development, and other health problems. If the followup evaluation identifies an abnormality, a referral is made to the parents and the primary care physicians.

Each year, approximately 5,300 infants from birth to one year of age are placed on the high-risk registry with the New Jersey Special Child Health Services Program because they have been identified as having special needs as a result of a handicapping or potentially handicapping condition or chronic illness. Parents of children entered on the high-risk registry receive a letter confirming this registration. The county case manager then contacts the parents to inform them of services which are available. Parents decide for themselves whether or not they want to take advantage of these services. Most children in need of early intervention services, however, are not identified through this system. They may be omitted from the registry at the physician's discretion, parents may choose not to followup, or more typically, the children experience normal birth and delivery and later are found to have developmental dysfunction.

EARLY INTERVENTION SERVICES IN NEW JERSEY

Early intervention programs are available in New Jersey for all children who need them, free of charge. There are 38 early intervention programs in New Jersey. Eligibility for these programs and services is based upon assessment which establishes either of the following: 1) The child has a condition that impairs or has a high predictability of impairing normal attainment of developmental milestones; and 2) The child has measurable delay(s) or disordered behavior as verified by assessment indicating that the child is functioning at least 25 percent below his/her chronological age in two or more developmental areas.

A definitive medical diagnosis is not required for a child to be eligible for the services of an early intervention program.

The professionals who provide services in early intervention programs represent the wide array of disciplines necessary to meet the complex therapeutic, educational, and social needs of the infant and family. This core team of professionals provides services to infants and families in an interdisciplinary manner. As team members work together, they are teaching and learning from one another as they provide intervention services by combining their expertise. All services to infants and their families in an early intervention program are delivered through this interdisciplinary team approach. These services include: identification and intake, screening, comprehensive evaluation, individual service program (ISP) development, intervention procedures, and followup.

A request for the services of an early intervention program must be made by the parent and signed before screening or assessment occurs. When the physician refers children and their parents to an early intervention program, it is important to include pertinent background information along with the reason for requesting service, diagnosis (if known), any previous evaluation, and actions to be taken. The role of the pediatrician or family physician in the assessment of the child suspected to be at risk of being delayed is vitally important as he/she may well be the only professional to help direct a family to an early intervention program in a timely fashion. To help determine referral to a special program, the physician may employ a developmental screening test to divide children into two categories—at risk and not at risk.

The child may be tested directly by the physician or a qualified staff member. Parental measures also may be used such as a parent interview or parent take-home observation checklist. If a child falls into the "at-risk" category after the primary screening, then the child should be referred to an early intervention program for more indepth developmental assessment. Screening provided by the early intervention program may be used by the physician if he/she is not sure whether the child is eligible for early intervention services. Following screening, if a possible developmental delay is indicated, children should be referred for a comprehensive evaluation. The comprehensive evaluation includes a health assessment and a developmental evaluation which addresses all areas of the child's growth. The cost of the health assessment generally is borne by the family. However, each program makes provisions to ensure that all families have access to this service. Most often the health assessment is performed by the child's personal physician and this information then is sent to the early intervention program.

The next step usually is a developmental evaluation, which consists of an appraisal of the child by the core team in the following areas: motor development, communication, sensory development, cognitive skills, social/emotional development, self-help skills, and feeding. The evaluation team consists of at least four professionals who collaborate on assessment procedures as well as the development of findings and recommendations. An ISP is developed by the core team in consultation with the parents once eligibility is determined. All of the activities in an early intervention program reinforce the role of the parents as primary caregivers and decision makers for their children. A major focus of the staff is to support the attachment between the infant and the family, and to strengthen the family's sense of competence in coping with the needs of their child.

New Jersey physicians are instrumental in ensuring that infants at risk are linked to necessary intervention services at an early age. Delaying intervention can be costly.

ROLE OF THE PHYSICIAN

The physician is a vital component to this service system as it exists in New Jersey. He/she often is the professional who has further ongoing face-to-face contact with these parents and their infants at risk of developmental delay. Informing parents that their child is suspected of having a handicap usually is not an easy task. If the parents themselves suspect that their child has a problem, then referral to an early intervention program for services most often proceeds with little resistance. It is a different matter when the parents do not suspect that their child has a problem or when they are having difficulty acknowledging it. Sensitivity yet persistence is encouraged in these situations. Assessment by an early intervention interdisciplinary team will help the parents understand how early intervention can help if delays are confirmed. Having a suspected delay checked out further is an important role of the physician monitoring the progress of infants. There will be instances where the physician may not suspect a problem with a child, but the parents are concerned that there may be one. One possibility to be considered is to give the parents the opportunity to evaluate their own child at home with a simple parent screening instrument. If parental concern persists after evaluating their child, referral to an early intervention program for further assessment either may confirm delays or ease worries on the part of the parent. It is important that parents be prepared before they visit the early intervention site. They should be informed that children with a wide range of problems attend such programs and that even children with less apparent problems may be enrolled.

In 1973, there were relatively few infants being served in the first early intervention programs in New Jersey. In 1985, approximately 3,000 infants will be served at no cost to parents in 38 state-funded early intervention programs. Physicians are a vital component in this service delivery system and their important role in early detection and referral of handicapped infants is vital. Physicians are instrumental in ensuring that infants at risk are linked to necessary intervention services at an early age. Delaying intervention is costly for the child, for the parents, and for the taxpayers. Beginning intervention early reduces these human and financial costs and is an essential step in the process of integrating individuals with special needs into the mainstream of society.

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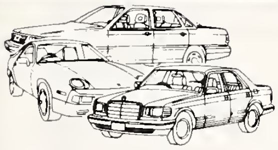

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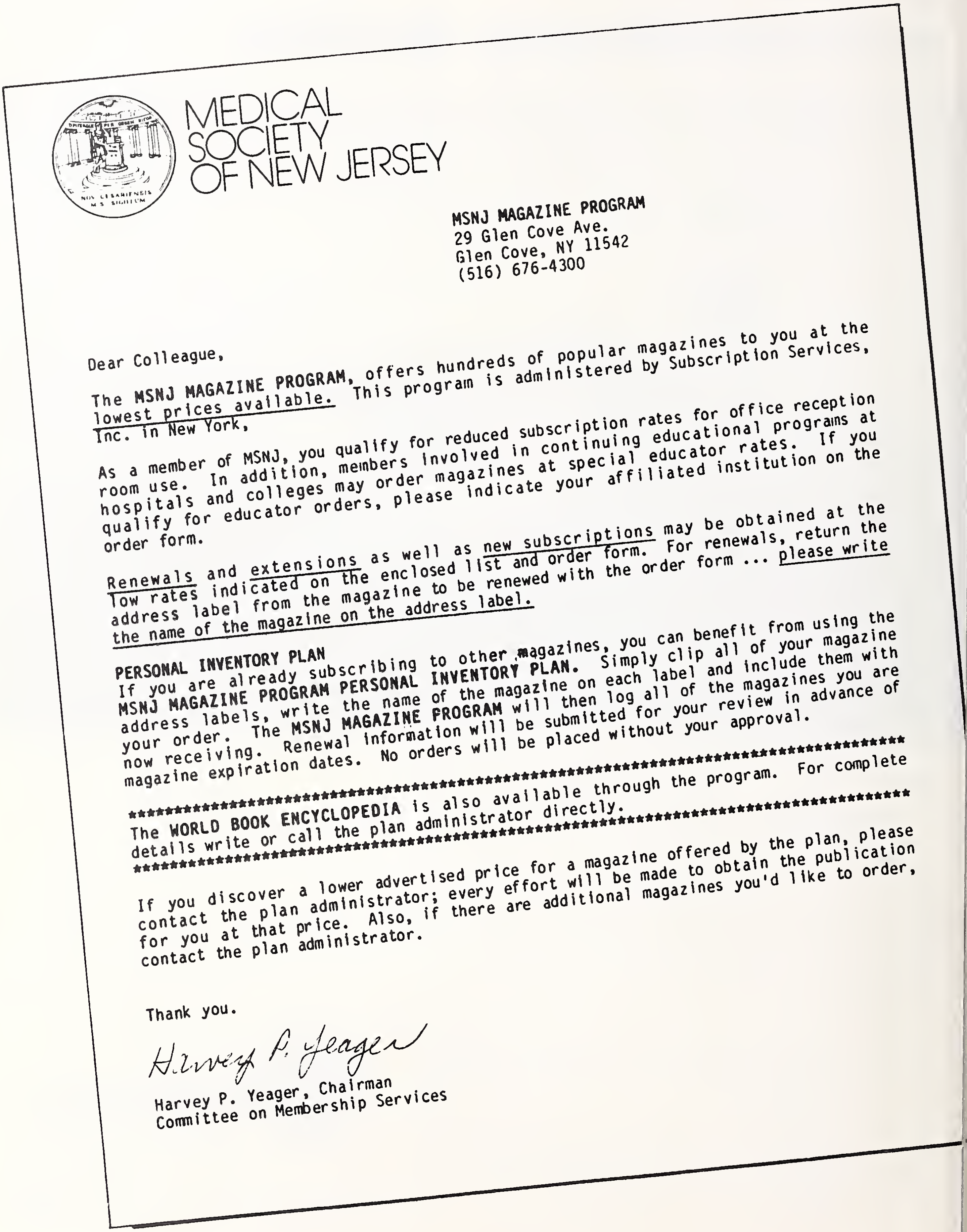

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CASE REPORT: SPONTANEOUS RUPTURE OF THE STOMACH

WILLIAM L. DIEHL, M.D., AND EUGENE L. WATKINS, M.D., MORRISTOWN*

Our patient had spontaneous rupture of the stomach with extensive necrosis of the greater curvature. Experience with this case of gastric perforation and a review of the literature reveal a high incidence of complications.

Gastric perforation may result from a primary cause such as trauma or ingestion of corrosive substances, but spontaneous rupture of the stomach is rare. The following case report documents such a problem with its diagnostic and therapeutic trials and tribulations.

CASE REPORT

A 36-year-old white female schoolteacher came to the Morristown Memorial Hospital Emergency Room because of severe abdominal pain of two days' duration. The pain grew worse and was accompanied by anorexia, fever, nausea, and vomiting. Approximately six hours before the abdominal pain started, she had ingested a moderately large meal of fish; but the total volume did not exceed 800 to 1000 cc.

The past medical history was negative except for precocious puberty of unknown etiology and she was in good health. She did not smoke or drink alcohol. Preadmission medication was medroxyprogesterone (10 mg daily during seven days of each cycle) for irregular menstrual periods.

On admission, hemoglobin was 16 gm/dl; Hct, 48%; WBC was 23,000 with 85% polys, 12% bands, and 2% lymphocytes; SMA-6, urinalysis, and clotting studies were normal. X-ray of the chest and abdomen showed free air under the diaphragm.

With a presumptive diagnosis of perforated duodenal ulcer, laparotomy was performed and necrosis of the fundus and greater curvature of the stomach with free perforation and generalized peritonitis were found. Resection of the necrotic third of the stomach along the greater curvature permitted a primary closure and the formation of a more tubular stomach. A splenectomy was performed due to the severity and extent of the inflammatory process. During the operation, she received 7,000 cc of crystalloid fluid and 1,500 cc of 5 percent plasma fraction.

The immediate postoperative period was disturbed by severe hypotension and metabolic derangements: arterial blood pH was 6.97; serum potassium was 1.4 mg/dl; and glucose was greater than 1,000 mg/dl. Chest x-ray showed pulmonary edema compatible with adult respiratory distress syndrome. A Swan Ganz catheter, arterial line, and endotracheal tube were inserted; her metabolic derangements were corrected gradually. Continued need for ventilatory support required a tracheostomy on postoperative day 8. Acute pancreatitis with serum amylase levels greater than 900 units also developed. Signs of sepsis continued with persistent fever and leucocytosis; on postoperative day 9, an abdominal ultrasound showed a left upper quadrant collection.

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A gallium scan confirmed a left upper quadrant abscess and a gastrografin study revealed free leakage of contrast material into the left subphrenic space. Surgical re-exploration disclosed a left subphrenic abscess extending behind the stomach remnant; it was drained and a perforation along the suture line of the stomach was repaired. Postoperatively, her respiratory status improved and she was weaned from the ventilator, but spiking temperatures and leucocytosis up to 46,000 continued. CT scan of the abdomen on day 16 after her second operation revealed a left subphrenic abscess and a right subhepatic fluid collection. A third laparotomy was performed to drain the recurrent abscess, and a small subhepatic hematoma. Cultures from the abscess grew *E. coli* and *Pseudomonus aeruginosa.* With large sump drains in situ, she was treated with tobramycin, clindamycin, and ampicillin. The patient gradually improved, but 8 days after her third operation, she developed a wound dehiscence with evisceration. Once again, the left upper quadrant cavity was explored surgically and irrigated but no frank abscess was found. A persistent gastrocutaneous fistula was managed conservatively with total parenteral nutrition (TPN) and enteral feedings through a jejunostomy tube inserted at the time of her second operation. She was finally discharged on hospital day 89 in good condition and able to tolerate an oral diet.

DISCUSSION

Perforation or rupture of the stomach, an infrequent occurrence, accounts for 0.9 to 1.7 percent of hollow organ perforations during blunt trauma to the abdomen.[1] It may develop spontaneously or as a complication of peptic ulcer disease.[2] Inadvertent or suicidal ingestion of corrosive acids commonly cause the most damage to the stomach as compared to the esophagus which, by contrast, most commonly is affected when alkaline substances are ingested.[3] In these cases, if the patient does not die from a perforation, peritonitis, or shock in the first day after the ingestion, delayed complications such as pyloric stenosis and delayed perforation are not uncommon.[3] However, erosion of the stomach has been reported after ingestion of alkali with sparing of the esophagus.[4]

Rupture of the stomach without antecedent trauma is even more rare. The mechanism, regardless of its etiology, often involves overdistention of the stomach. It is postulated that after a certain degree of distention, the gastroesophageal junction forms a valve mechanism impeding the exit of air and food to the esophagus, but permitting its entrance into the stomach. This is illustrated dramatically in a recent case report by Goff and Norton whose patient developed severe abdominal distention after a copious meal and was unable to vomit or to have a nasogastric tube placed.[9] The increased intra-abdominal pressure caused loss of lower extremity pulses due to pressure on the aorta in addition to cardiorespiratory arrest. At laparotomy, she was found to have a gastric tear. The distensibility of the greater curvature permits it to accommodate a larger volume, but the lesser curvature is more fixed and less distensible, and is a more frequent site of perforation as in the Mallory Weiss syndrome when acute pressure is applied.[5] However, if the stomach is able to tolerate this distention, and the intraluminal pressure exceeds the venous pressure and the venous drainage is compromised, then perforation is possible at any part of the stomach.[6] Ingestion of sodium bicarbonate and severe bouts of vomiting,[3] and less common conditions such as seizures, oxygen insufflation, use of atropine eye drops, and intubation for anesthesia have been described as etiologic causes for spontaneous rupture of the stomach. Gastric perforation also may take place with mouth-to-mouth ventilation during cardiopulmonary resuscitation.[8]

Our patient was interesting because a clear etiology for the gastric necrosis could not be found. The large meal she ingested obviously is suspect though it was not as excessive as has been described in those cases with associated gastric perforation; there, volumes of four liters have been exceeded.[5] She had not ingested alkaline or acid substances and had not sustained any type of injury to the abdomen. As mentioned, hormonal ingestion has been reported in association with small bowel perforation but not with gastric perforation.[7]

SUMMARY

Spontaneous gastric perforation from entities other than peptic ulcer disease is rare, but must be considered in the differential diagnosis of free intraperitoneal air. As noted in this report and others in the literature, these patients frequently follow a stormy postoperative course. The attending surgeon, and colleagues, faced with such a patient, should be prepared for multiple severe complications.

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COMMENTARY: RECOMBINANT DNA TECHNOLOGY AND BIOLOGICAL WARFARE

DONALD B. LOURIA, M.D., NEWARK*

Some of the most spectacular biomedical technological advances are being utilized not only to help mankind but also for potential use in warfare. This should be of major concern to the medical profession.

Five years ago, I became concerned about the possibility of military misuse of recombinant DNA technology to create microbial monsters. I assumed that once the technology was available, the fears of the governments of the Soviet Union and the United States would drive each to manufacture gruesome biologic weapons. To focus attention on this issue, I wrote an article, published in *Futurist*, in which I "created" a hypothetical lethal organism using recombinant DNA techniques.[1] The organism required certain characteristics: it had to be a normal constituent of the intestinal tract so its introduction into a population would not arouse suspicion for a substantial time; it needed the capacity to dislodge endogenous intestinal flora; it had to possess broad resistance to antibiotics; and it should contain one or more lethal toxins.

It is not hard to construct such an organism. I chose *Escherichia coli*, the most prevalent gram-negative aerobe in the intestinal tract, and hyperpiliated it; that should be easy since piliation is under genetic control. Hyperpiliation would allow a modified *E. coli* to displace endogenous intestinal *E. coli*. Next, plasmids to confer resistance to all known antibiotics would be added. The last step is to add one or two toxins that are lethal and for which there is no antidote. I selected four for potential uses: ricin from the castor bean; aconitine from wolfsbane; tetrodotoxin from the puffer fish; and trichothecene toxins from the fungus *Fusarium sporotrichioides*. Each has marvelous lethal properties.

There were two major criticisms of my article: the microbe conjured up was not easy to produce and we might not be technically able to do it for at least a few years; and nobody would bother creating such an organism because we already had good biological warfare weapons (such as the organisms causing anthrax, botulism, tetanus, and diphtheria).

The following has happened since that article appeared:

1. The Department of Defense (DOD) has supported at least a dozen projects to produce vaccines by genetic recombination techniques. They insist that such vaccines are for defensive purposes only, but surely it is evident that if you develop a vaccine for a life-threatening disease, by subterfuge you then can immunize your own people and turn a defensive weapon into a potentially offensive weapon. The latter depends on the proviso that your adversary does not suffer from that disease in endemic form (thus producing herd immunity) and does not have the vaccine. The DOD projects must scare the Soviets, especially since there is at least talk of developing vaccines by recombinant

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DNA techniques against viruses of Lassa fever, Marburg disease, Ebola fever, and equine encephalitis.

2. Researchers at Harvard University requested permission to put diphtheria toxin into *E. coli.* Some people might say we do not need diphtheria toxin made in bulk by genetically manipulated *E. coli.* The project initially was rejected by the Recombinant DNA Advisory Committee of the National Institutes of Health (NIH) which has purview over government-funded recombinant DNA research. The rejection was reversed on appeal in 1983 on the grounds that the scientific value of the study for potential cancer therapy outweighed any concerns. Nobody apparently gave much consideration to how the Soviets would interpret this well-publicized investigation.

3. In 1984, the columnist Jack Anderson wrote several columns saying that the Pentagon had information that suggested strongly the Soviets might be moving ahead with biologic warfare programs. The "secret" CIA analysis had no definitive proof but lots of reasons for major concerns.

4. With what, at the very least, was lack of candor, the Pentagon snuck through Congress a revision of already approved military appropriations to permit a multimillion dollar renovation of a site in Utah[2] that had been used for chemical warfare studies. The DOD wants a sophisticated biologic and chemical warfare unit established for "defensive" purposes only.

Of course, what actually goes on in the Utah facility or in other DOD or CIA projects is to be kept secret. Whether the investigations include potential attack weapons is left to our imagination and the paranoia of the Soviet hegemony.

It is unclear who started the burgeoning competition in biological warfare. It does not really matter. What is important is that the two super powers, fearful of each other, and possessing increasingly sophisticated technology, are engaged in another repulsive adventure into methods for killing entire populations. Thus, they are transmogrifying the advances in the biologic sciences that could be used for the benefit of mankind. They are doing so in contravention of at least the spirit of signed treaties and worldwide perceptions of what is acceptable in man's unending determination to destroy other members of the species.

This pursuit of microbial weapons for warfare purposes using recombinant DNA techniques impinges directly on a major area of interest of the medical community. It, therefore, is our obligation as a profession to speak out forcefully. We cannot, as we so often have done in the past, withdraw into a solipsistic medical cocoon. We have credibility in this area and we must play a role in formulating policy. We could, for example, insist that:

1. All such studies for "defensive" purposes be open to scrutiny, including scrutiny by other nations;

2. A Soviet scientific observer be invited to participate in every project involving recombinant DNA technology that might be perceived as either an offensive or defensive biological warfare weapon; the Soviets would be expected to reciprocate.

3. Any vaccine that is produced be made available to any country in the world either as a vaccine or in the form of the genetically altered microbial vaccine factory.

There are other suggestions that could be made. And, there is much we as a nation could do to reduce international tensions so that biologic warfare "defensive" efforts could be abandoned altogether or greatly reduced.

What we cannot afford is insouciance. The various American medical organizations should speak out now or we may find that while our heads were turned, our world disappeared.

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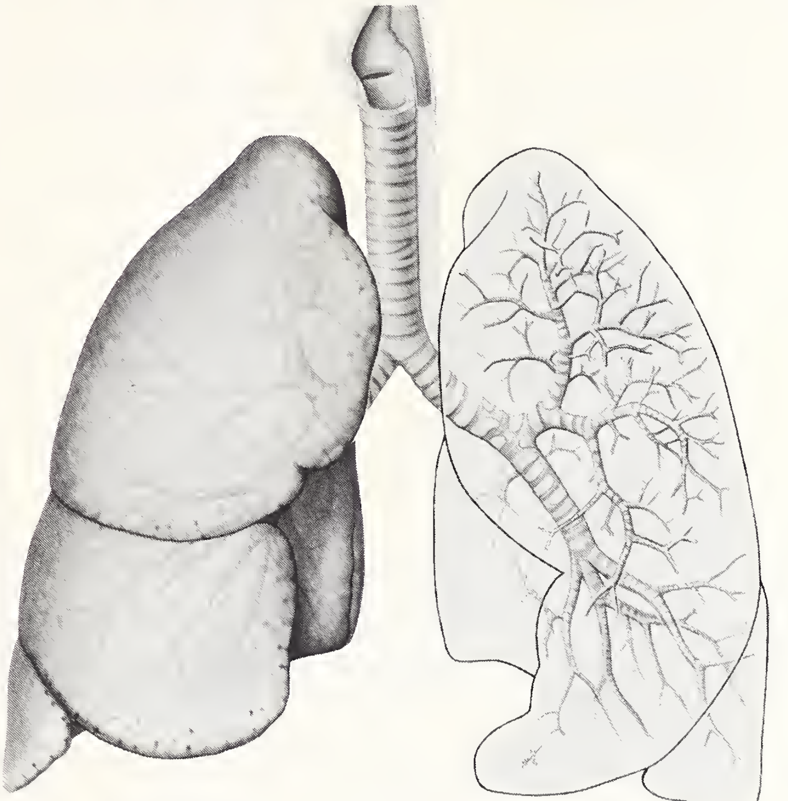

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OPINION: NEW ISSUES IN THE CARE OF THE MENTALLY ILL

GEORGE A.. ROGERS, M.D., CAMDEN*

Our deinstitutionalized mentally ill cannot be treated properly because of inadequate commitment laws. Physicians should be knowledgeable of the current problem and supportive of change.

Members of the Medical Society of New Jersey should be aware of pressing new issues in the care of persons who are severely mentally ill. Some years ago, with the Donaldson decision in Florida,[1] the court decided that patients could not be detained in a mental hospital against their will, unless they were a "danger to themselves or others." Since that time, a number of significant court decisions have limited the conditions under which a patient can be hospitalized involuntarily and strengthened the right of a patient to refuse treatment, even though committed to a mental institution.[2,3] With this attention to the civil rights of patients has come a thrust by state mental hospitals toward "deinstitutionalization."

The result has been constructive in some instances and, in others, quite tragic. It has led to "bag ladies" and street people who continue to inhabit large cities and their jails. The "catch 22" in mental illness has been that the illness itself frequently deprives the patient of the insight and judgment required to acknowledge the illness and the need for medication. Thus, 50 percent of deinstitutionalized patients are noncompliant as to medications following discharge from the hospital.[4] Unfortunately, the less medication the patient takes, the less likely he will resume medication; the more mentally ill he becomes, the more suspicious he becomes of mental health personnel; and, therefore, he is less likely to seek help. Current laws prohibit such patients from being treated or hospitalized involuntarily unless they are an immediate danger to themselves or others. We do not allow delirious and confused medical patients to walk out of our general hospitals. Certainly, confused mentally ill patients should be given the same concern.

These strict laws have led to several dramatic tragedies. A North Jersey physician, incompletely recovered from a serious depression, was allowed to sign himself out of the hospital because immediate and severe danger to self could not be proved; he killed himself. Another example is a chronically psychotic patient, residing in a boarding home in southern New Jersey, who got into an altercation with his landlady and killed her. The patient had been cooperative and nonviolent when released to the boarding home, but there was no means of insuring that he would continue to take medicine. He discontinued it and became more psychotic; tragedy resulted.

These dramatic examples of our inability to treat patients until they have actually done something demonstrating "danger to themselves or others" are not the most important problem, however. The most important and frequent problem is the psychotic patient who, while treatable, remains in the community in a chronic state of confusion.

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The following is a quote from a paper by Darold Treffert, who reports a letter from a daughter about her mother.[5] The daughter described her fears and frustrations about the care of her mentally ill mother, who had been ill for more than 15 years and required numerous hospital admissions for psychiatric problems. As reported by Treffert, the daughter wrote to her legislator:

"Two weeks ago, my husband and I went to the district attorney's office in a last resort effort to obtain psychiatric help for my mother, who is mentally ill. Much to our frustration, he informed us that, due to recent changes in state law, mentally ill persons cannot be treated unless they request treatment, are suicidal, dangerous to others, or unable to take care of themselves. The latter is applicable, we are told, only in extreme cases, such as refusing to eat for prolonged periods or sleeping "in the gutter" in subzero weather.

"For the past year, Mother has lived at a residential center. She was doing quite well. However, she began to skip her medications, becoming increasingly agitated and bizarre in her behavior. She was 'discharged' from the residential facility because she was unmanageable, refusing to take her medicine or admit herself to a hospital. The ensuing weeks have been, in simple terms, terrible, traumatic, and exasperating for my sister, my husband, and myself, and I am certain, for my mother, as well.

"She immediately spent $60 to $70 and turned up at our apartment without a cent and without a place to go. We convinced her to go to the hospital, and she was admitted voluntarily. She signed herself out. At midnight, we were called by the police. She had ordered a lobster dinner, and left the restaurant without paying. The police took her back to the center, but since she refused to sign herself in, I took her to the counselor for the Family Protective Team. She convinced my mother to enter a private hospital, where she signed herself out. She had 50 cents to her name. The police called again reporting that mother again had ordered a dinner for which she did not pay. They took her again to the center where she refused even to see a doctor.

"That afternoon she phoned me from the cemetery, insisting that my father was out of his grave and causing her a lot of trouble. My mother was arrested for loitering. She appeared unkempt and dirty and was carrying a bag full of garbage. The jail called, concerned by the fact that this was mother's third arrest for disorderly conduct in the week. The matron saw her need for psychiatric care, as did the arresting officer and the municipal judge. The case went to circuit court, but the public defender wanted no part in helping someone "put their mother away." As a result, she again is wandering the city streets, and sleeping in bus depots.

"We have been advised by attorneys and social workers that our only alternatives are: 1) temporarily to disown her so that she will have no one at all to turn to; 2) to threaten her with commitment in hopes that she will sign herself in instead; or 3) to wait until the police pick her up for a criminal offense, i.e. theft, and she can be taken to criminal court where the judge readily would recognize her as mentally ill and send her instead to a hospital under court order.

"We cannot treat mental illness by denying its existence. While psychiatric hospitals certainly are not the most pleasant situations, I have seen my mother recover when treated there and be discharged to lead a more peaceful, happy life. I do not feel that my mother is receiving just treatment or any benefit from a law that leaves her to wander the streets mentally disorganized and physically run down. I must mention also the mental anguish, frustration, and severe emotional strain shared by our family and mirrored in the many law enforcement officers and other officials with whom we have come in contact. We are told to disown our own mother in order for her to receive proper care. Is this preservation of anyone's right to life, liberty, or the pursuit of happiness? My mother's illness is of a chronic nature. Must we anticipate yearly repetitions of this hopeless episode?"

Most psychiatrists practicing in the community regularly see similar problems, although perhaps not quite as graphic nor as well described. These obviously are ill patients who can respond to treatment if it can be given to them, as was the case as this daughter described her mother.

At present, a legislative committee is considering the formulation of new commitment laws which will place both the "gravely disabled" and the "obviously ill patients in need of treatment under the court's jurisdiction and treatment."[6] With encouragement and support from physicians and concerned citizens, New Jersey can have a law which protects the civil liberties of the mentally ill, while at the same time protecting their right to treatment.

Clearly, improvements in the laws can enable many patients to lead a normal life and be productive citizens. The addition of the "gravely disabled" and "obviously ill" concepts to our state's involuntary treatment laws will help a great many individuals who are living only a half-life. The addition of these classes of treatable illnesses can be done with proper judicial safeguards. The patients' civil rights need not be violated. Certainly, living in the darkness of insanity is a loss of liberty of the worst kind. Citizen and professional pressure for legal change will relieve this problem.

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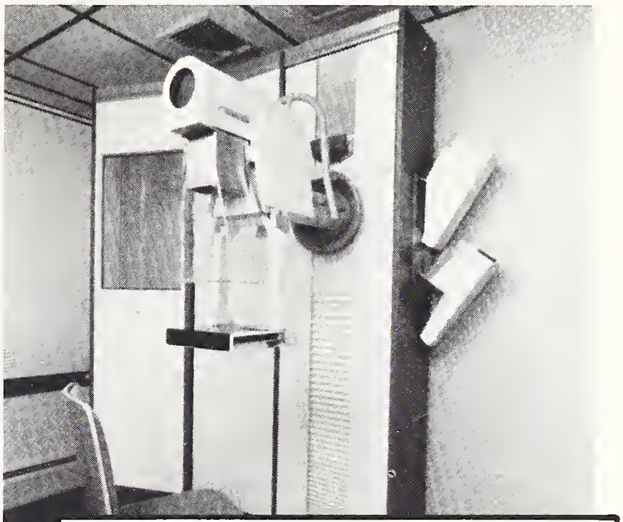

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GOLDEN MERIT AWARD RECIPIENTS

At ceremonies held on May 2, 1986, during the 220th Annual Meeting of the Medical Society of New Jersey, at the Americana Host Farm Resort, Lancaster, Pennsylvania, the following received MSNJ's Golden Merit Award indicating they held the degree of Doctor of Medicine for 50 years.

Atlantic County

Irving E. Braverman, M.D.	Middlesex '36
Orazio J. DeSantis, M.D.	St. Louis '36
Leonard S. Ellenbogen, M.D.	Creighton '36
Amerigo Louis George, M.D.	Bologna '36

Bergen County

Eugene Andrick, M.D.	Temple '36
Leonard Brown, M.D.	St. Louis '36
Edward Calabrese, M.D.	Hahnemann '36
Joseph J. Coughlin, M.D.	Georgetown '36
Joseph Ferrante, M.D.	Bologna '36
William C. Giordano, M.D.	New York University '36
Paul L. Grosner, M.D.	Vienna '36
Edward F. Grueninger, M.D.	Long Island '36
Robert H. Ringewald, M.D.	Georgetown '36
Edo Joseph Salva, M.D.	Hahnemann '36
Michael Shenkman, M.D.	Belgium '36
Mendel Silverman, M.D.	Bern '36
Joseph J. Speranza, M.D.	Bologna '36
Frank Spiaggia, M.D.	Palermo '36
Charles G. Stetson, M.D.	Cornell '36

Burlington County

Edward C. Jennings, M.D.	Temple '36
Perry S. MacNeal, M.D.	Michigan '36

Camden County

Joel B. Cunningham, M.D.	Arkansas '36
Paul G. Ebner, M.D.	Jefferson '36
Harry E. Fridrich, M.D.	Hahnemann '36
Herbert F. Johnson, M.D.	Pennsylvania '36
John S. Klinger, M.D.	Bologna '36
Harry A. Pinsky, M.D.	Hahnemann '36

Cumberland County

Leonard G. Scott, M.D.	Meharry '36
Marvin Nathaniel Solomon, M.D.	Bern '36

Essex County

Frank J. Albano, M.D.	Rome '36
John E. Barrett, M.D.	Hahnemann '36
Otto S. Baum, M.D.	Harvard '36
Gerald J. Booken, M.D.	Temple '36
Benjamin B. Burrill, M.D.	Duke '36
Jack Chernus, M.D.	Glasgow '36

Joseph D. Cuono, M.D.	New York Medical '36
Abraham L. Dear, M.D.	Western Ontario '36
James H. DeGerome, M.D.	Long Island '36
Samuel Diener, M.D.	Michigan '36
Edward P. Duffy, Jr., M.D.	Georgetown '36
Samuel J. Fortunato, M.D.	Jefferson '36
Benjamin Gencher, M.D.	Queen's '36
William E. Grant, M.D.	Long Island '36
Eugene Greenwald, M.D.	New York University '36
William Greifinger, M.D.	Maryland '36
Robert J. Gross, M.D.	Zurich '36
Marie Kaney, M.D.	Vienna '36
Irwin Kimche, M.D.	Genoa '36
George P. Koeck, M.D.	New York University '36
Milton Lilien, M.D.	Iowa '36
Saverio A. Monaco, M.D.	Hahnemann '36
Thomas D. Monte, M.D.	Long Island '36
Jaroslaw Myndiuk, M.D.	Poznan '36
Salvador Dante Pentecost, M.D.	Maryland '36
Felix H. Piegari, M.D.	Bologna '36
Herman I. Roseman, M.D.	Geneva '36
Sam Rosenbaum, M.D.	New York Medical '36
Lewis E. Savel, M.D.	New York Medical '36
Harold Schwartz, M.D.	Vienna '36
Irvin I. Taitz, M.D.	Basel '36
Albert B. Tucker, M.D.	Pennsylvania '36
Max S. Yablonsky, M.D.	New York University '36
Jack S. York, M.D.	Bellevue '36

Gloucester County

William Stephen Serri, M.D.	Hahnemann '36
Ezra Bernard Sirotta, M.D.	Hahnemann '36

Hudson County

Francis Leo Boyle, M.D.	Marquette '36
Vita Francis Cangemi, M.D.	Georgetown '36
Robert Allison Cosgrove, M.D.	Cornell '36
Anton Cziraky, M.D.	Vienna '36
Silverino V. DeMarco, M.D.	Boston '36
Irving Dershewitz, M.D.	Berlin '36
Harry Hauptman, M.D.	Washington-St. Louis '36
Harold Brooklin Schwartz, M.D.	Louisville '36
Louis Frederick Sciarrillo, M.D.	Rome '36
Paul Francis Sinclair, M.D.	Howard '36
Harry Lewis Stark, M.D.	St. Louis '36
Joseph Edward Zbar, M.D.	Queen's '36

Hunterdon County

Vladimir Ctibor, M.D.	Maryland '36
John F. Fritz, M.D.	Pennsylvania '36
Edwin V. Olmstead, M.D.	Pennsylvania '36

Mercer County

Joseph Robert Burns, M.D.	Temple '36
Herbert Marx Wolff, M.D.	Jefferson '36
Kurt Zeltmacher, M.D.	Prague '36

Middlesex County

Sigmund J. Clayman, M.D.	Zurick '36
Mortimer Cowen, M.D.	Zurick '36
Bernard Friedenthal, M.D.	Bern '36
George John Kohut, M.D.	Budapest '36
Philip J. Kunderman, M.D.	Cornell '36
Reuben Levinson, M.D.	Edinburgh '36
Thomas F. McLaughlin, M.D.	Georgetown '36
Michael Mendelson, M.D.	Vienna '36
Louis Arthur Perillo, M.D.	New York Medical '36
George Racz, M.D.	Budapest '36
Norman Reitman, M.D.	New York University '36
Eugene Revitch, M.D.	Paris '36
John Albert Smith, M.D.	Jefferson '36
Sydney F. Smith, M.D.	Bellevue '36
Mathilda R. Vaschak, M.D.	Woman's Medical '36

Monmouth County

Sidney M. Hodas, M.D.	Jefferson '36
Joseph LoBuono, M.D.	Rome '36
Victor Siegel, M.D.	George Washington '36
Vincent M. Whelan, M.D.	Harvard '36

Morris County

Oscar H. Cohen, M.D.	Jefferson '36
Amedeo Esposito, M.D.	Rome '36
Guy Henry Laudig, M.D.	Harvard '36

Ocean County

Jacob M. Goldstein, M.D.	Innsbruck '36
Willis B. Mitchell, M.D.	Cornell '36

Passaic County

Daniel Bergsma, M.D.	Yale '36
Alphonse Brancone, M.D.	Naples '36
Alfred Bronner, M.D.	Vienna '36
Robert Floody, M.D.	Albany '36
Seymour P. Jaslow, M.D.	New York University '36
Theodore R. Keith, M.D.	Temple '36
Nadhim E. Meer, M.D.	Iraq '36
Jacob R. Schwartz, M.D.	Vienna '36
Blanka Sender, M.D.	Cracow '36
Thomas Siciliano, M.D.	New York Medical '36
Louis Small, M.D.	Jefferson '36
Morris H. Stern, M.D.	Maryland '36
Abram Vermeulen, M.D.	Northwestern '36
Stephen L. Weisman, M.D.	Rush '36

Sussex County

Frank Herbert Lushear, M.D.	Columbia '36

Union County

Seymour Ralph Deehl, M.D.	Maryland '36
Warren P. Dochtermann, M.D.	Rochester '36
George L. Erdman, M.D.	Jefferson '36
Samuel M. Hoch, M.D.	New York University '36
A. Starr Ingram, M.D.	Emory '36
Samuel D. Kaplan, M.D.	Pennsylvania '36
Alexander Lewis, M.D.	Perugia '36
Estelle T. Milliser, M.D.	Chicago '36
Roland Drew Roecker, M.D.	New York Medical '36
Michael William Spirito, M.D.	George Washington '36
Jerome Marshall Wolff, M.D.	Pennsylvania '36

DOCTORS' NOTEBOOK

Trustees Minutes; UMDNJ Notes; MSNJ Auxiliary; AMNJ Report; Physicians Seeking Location in New Jersey

Trustees' Minutes April 13, 1986

A regular meeting of the Board of Trustees was held on Sunday, April 13, 1986, at the Executive Offices in Lawrenceville. Detailed minutes are on file with the secretary of your county society. A summary of significant actions follows:

Report of Executive Director . . .
(1) Smoking Ban in MSNJ Building . . . Directed that the following recommendation be enforced:

> That a no-smoking policy for all employees, members, guests, and committee functions within the Medical Society of New Jersey headquarters, effective June 1, 1986, be adopted.

(2) Uniform Birth Record Project . . . Approved the following recommendation:

> That the Medical Society of New Jersey participate in the Uniform Birth Record Project.

(3) Budget Reconciliation Bill . . . Noted that the Medicare fee freeze has been extended through April 30, 1986, for all physicians; after that date, the fee freeze will remain in effect for nonparticipating physicians through December 31, 1986.

(4) HCFA Medicare Claims Delay . . . Directed that an appreciative letter be sent to Sen. Frank Lautenberg for his efforts in protesting the delay in payment of Medicare claims instituted by the Health Care Financing Administration.

(5) Advisory Opinion on Involvement of Attorney General's Office in State Board Disciplinary Proceedings . . . Approved the following recommendation:

> That the Board authorize Legal Counsel to file an amicus brief on behalf of the Medical Society of New Jersey, supporting the position of the Advisory Committee on Professional Ethics.

(6) MSNJ Paid Memberships . . . Noted that as of April 10, 1986, there were 6,600 paid members.

(7) Medical Malpractice . . . Noted that Mr. Maressa will testify before an Assembly Insurance Committee Hearing on Tort Reform.

UMDNJ Report . . .
(1) National Residency Matching Program . . . Noted that nearly 90 percent of UMDNJ seniors received their first or second choice in graduate medical education programs.

(2) Budget Reconciliation Act . . . Noted Dr. Bergen's comments on direct and indirect cost cuts in the Budget Reconciliation Act.

(3) Residency Programs . . . Noted Dr. Bergen's concern about the number of residencies in New Jersey.

(4) Malpractice Cases in Teaching Programs . . . Noted that Dr. Bergen agreed to participate in a proposed meeting to discuss teaching programs related to malpractice cases involving a practice within the Attorney General's office.

NJ Hospital Association . . .
(1) Uncompensated Care Legislation . . . Noted that the NJHA is collaborating with the New Jersey Department of Health to achieve the compromise language required to establish a trust fund and pool to pay for hospital uncompensated care.

(2) Tax Reform Legislation . . . Noted that NJHA is lobbying to have hospitals exempted from caps on tax exempt bonds on a per capita basis.

(3) Directors' and Officers' Liability Coverage . . . Noted that liability coverage of hospital directors and officers is a serious problem since the Midlantic Insurance Company's descent into Chapter 11.

MSNJ Resident Association . . . Approved the following recommendation:

> That the MSNJ Resident Association be granted representation in the House of Delegates.

Committee on Biomedical Ethics . . . Noted that the Committee is rewriting the guidelines for a living will with the focus on assuring protection for physicians attending terminally ill patients.

Council on Legislation . . . Approved all the positions on current legislation recommended by the Council (page 359), except for S-600 which was changed to Active Opposition.

Committee on Annual Meeting . . . Voted to reject the Committee's recommendation to hold the 221st Annual Meeting at Loew's Glenpointe Hotel and to hold it at Host Farm Resort from April 30 to May 3, 1987; also, accepted the recommendation to hold the 222nd Annual Meeting at Bally's Park Place Casino Hotel from May 19 to May 22, 1988.

Committee on Finance and Budget . . . Approved the following recommendations:

> 1) That the budget for the fiscal year beginning June 1, 1986, and ending May 31, 1987, in the amount of $3,124,000, with $2,321,000 to be raised through member assessments, be adopted.
> 2) That the 1987 assessment be set at $330 per regular dues-paying member (1986 assessment—$295).
> 3) That the 1987 assessment be set at $40 per member for affiliate members (no longer practicing in New Jersey) (no change).
> 4) That the 1987 assessment for associate members (interns-residents nonlicensed in New Jersey) and licensed residents provided the individual is in a residency program entered upon within a reasonable time after his or her graduation from medical school be set at $25.
> 5) That the 1987 assessment be set at $10 per student for medical students (no change).

Committee on Medical Education . . . Approved the following recommendation:

> That the Medical Society of New Jersey cosponsor a conference with the Association for Hospital Medical Education of New Jersey on the funding of graduate medical education. (Cosponsorship does not entail a financial contribution.)

Joint Meeting of Special Committee on Long-Range Planning and Development and Committee on Revision of Constitution and Bylaws . . .

(1) Referrals from Board of Trustees . . .

(a) Nominations of Candidates for Positions on AMA Delegation . . . Noted a subcommittee was formed to review the change in the nominating procedure for AMA Delegate positions.

(b) AMA Delegation Officers . . . Postponed discussion on whether the President of MSNJ should appoint the chairman and vice-chairman of the AMA delegation.

(2) Report of Subcommittee To Review Nominating Committee Structure . . . Voted to oppose the following recommendation:

That councils and standing committees of the Society shall be removed from the jurisdiction of the Nominating Committee and the House of Delegates, and members of all the Society's councils and committees shall be appointed by the President, with the concurrence of the Executive Committee.

(a) Functions and Procedures of Nominating Committee: Unanimously approved the following recommendation:

That the Nominating Committee shall consider candidates recommended by component societies, as well as any member of the Society who expresses an interest in being considered as a candidate for office.

Approved the following recommendation:

That the Nominating Committee shall conduct personal interviews of each prospective nominee for the positions of officers, trustees, judicial councilors, and AMA delegates and alternate delegates before candidates are placed on the Nominating Committee ballot.

And, voted to reject the following two recommendations:

1) That the Nominating Committee shall consist of seven members: Immediate Past-President of the Society, who will serve as chairman; one member elected by and from the Board of Trustees; and one member from each of the five judicial districts.

2) That each year, on an alphabetically rotating basis, one county in each judicial district shall have the elective right to select the nominating committee member. At the annual business meeting of the designated county, a slate of two or three candidates for the Nominating Committee position will be drawn from the floor. The names of the candidates will be placed on a ballot to be submitted to each member of the designated county. The Medical Society of New Jersey will be responsible for printing, mailing, and tabulating the ballots. The candidate receiving the most votes will be the elected nominating delegate.

(b) AMA Delegation Officers . . . Approved the following recommendation:

That the Chairman and Vice-Chairman of the AMA Delegation be appointed annually by the President, with the concurrence of the Executive Committee. Tenure of office shall be limited to six years.

Special Committee on Long-Range Planning and Development . . .

Agreed to take the following suggestions under advisement:

1) Efforts should be made to streamline and expedite the application process for new members. It was felt that requiring physicians to attend county medical society meetings prior to election is cumbersome and delays the process. Elimination of this requirement might be considered. The personal interview is essential; however, it was felt it would be more expedient to have a member of the county society conduct the interview at an appropriate hospital site.

2) The president of the medical staff of each hospital should be on local councils. The hospital is the focal point for physicians; county medical societies should have a direct tie with each hospital within the county.

3) An appropriate newsletter, issued at the state level, dealing with socioeconomic issues should be distributed to all practicing physicians in the state. This would serve to inform members as well as nonmembers of current issues affecting the medical community and make them aware of the efforts and accomplishments of the Medical Society of New Jersey; it also might serve to encourage nonmembers to join the Society. (The Committee noted that the Ad Hoc Committee on Health Care Reimbursement Policies has recommended the issuance of a similar periodical, on which action is pending by the Board of Trustees.)

4) Recruitment and retention of members should be on a peer-to-peer basis and should be structured through the hospital medical staffs. Active recruitment at the hospital site should be pursued. Hospitals are recognized as the power base of the future and the involvement of the hospital medical staffs and physician-to-physician contact at that level is considered essential.

5) An attempt should be made at the state level to organize conjoint services especially in those counties without full-time staffed offices . . . Because of budgetary and staff limitations, members in these areas are not always provided with necessary services as quickly and efficiently as today's environment requires.

Committee on Medicaid . . . Approved the following recommendation:

That the Medical Society of New Jersey join the Division of Medical Assistance and Health Services in requesting an increase in the present Medicaid physician reimbursement fees.

Committee on Medical Aspects of Sports: Approved the following recommendation:

That the Medical Society of New Jersey strongly urge the maintenance of an adequate physical education program in New Jersey schools with no reduction in the present graduation requirements.

Peer Review Oversight Committee . . . Approved the following recommendation:

That the Medical Society of New Jersey request the State Board of Medical Examiners to adopt a regulation requiring all physicians doing chart review for either reimbursement or quality purposes to:

(a) Sign, type, or print their name in a legible fashion; and

(b) Sign a written certification that they

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have read the patient's chart being reviewed before making their decision.

Ad Hoc Committee on Health Care Reimbursement Policies . . . Approved the Committee's suggestion that a separate communication on socioeconomic issues facing physicians be sent periodically to the membership, and referred the implementation of this suggestion to the President and his staff.

Statewide HMO/IPA Feasibility Committee . . . Noted that after a lengthy discussion of all the factors and arguments in favor and against MSNJ forming a statewide HMO/IPA, the following was accepted:

a) That the Society should contract for an alternate delivery system implementation study; and
b) That, providing the study warrants proceeding to implementation, the Medical Society of New Jersey, with the best interest of the patient, quality health care, and physician control in mind, form an alternate health care delivery system.

New Business . . .
Uniform Preadmission Certification Guidelines . . . Noted that a Resolution promulgating a uniform set of guidelines for all third-party payers or insurers in the matter of preadmission certification and review will be presented to the 1986 House of Delegates.

UMDNJ Notes

Stanley S. Bergen, Jr., M.D.
President

The names Robert Wood Johnson and Johnson & Johnson have long been synonymous with health care philanthropy in New Jersey. The man and the company he built have played an unparalleled role in supporting health care research and delivery throughout the state. We, therefore, are very proud that Johnson & Johnson has chosen to mark its 100th anniversary by making UMDNJ the largest beneficiary of the company's $7.5 million New Jersey Major Gifts program.

The largest portion of the $3.25 million commitment to the University will go to the magnetic resonance imaging center being constructed in New Brunswick. Johnson & Johnson will donate $300,000 in cash and $1.4 million worth of equipment to the University for use at the center, which is to be operated by New Brunswick Affiliated Hospitals, Inc., a consortium including Middlesex General-University Hospital and St. Peter's Medical Center. Both hospitals have major teaching affiliations with UMDNJ-Rutgers Medical School. The new center will allow the hospitals to use state-of-the-art diagnostic techniques in providing patient care, while serving as an excellent center for the research and educational programs of UMDNJ.

The other components of Johnson & Johnson's gift to UMDNJ, which will be funded over a five-year period, include:

- A $550,000 grant to help expand cardiovascular research at the University's New Brunswick/Piscataway campus, with the focus on hypertension and atherosclerosis.
- A $500,000 grant for a fund to underwrite basic research projects to address serious health problems affecting New Jersey.
- A $500,000 grant to provide scholarships for minority medical students. We will use the scholarship money to support our long-standing commitment to minority medical education. UMDNJ already provides a medical education to a large percentage of minority students. We are proud of that record and grateful to Johnson & Johnson for supporting it.

Aside from the grants directly to the University, Johnson & Johnson has donated $400,000 to the Center for Molecular Medicine and Immunology, an independent cancer research center working closely with the University and located on our Newark campus. The money will be used to support the center's research involving the use of antibodies to track and treat cancer.

Since UMDNJ was founded in 1970, Johnson & Johnson and the Robert Wood Johnson Foundation have been the largest financial benefactors of the University, aside from the state government itself. Their support has been so generous and so vital that the University has chosen to rename one of its six schools, the UMDNJ-Rutgers Medical School, for the late General Johnson. As of July 1, the school will be known as the UMDNJ-Robert Wood Johnson Medical School. Middlesex General-University Hospital, the primary teaching hospital for the school, on the same date will be renamed the Robert Wood Johnson University Hospital. The school's name change also should end the historical confusion between Rutgers Medical School and Rutgers-The State University; the two institutions have not been affiliated since 1970.

MSNJ Auxiliary

Mrs. John W. Holdcraft
President

Mrs. Grace Holdcraft is the newly elected President of the Medical Society of New Jersey Auxiliary. Born in Cleveland, Ohio, she attended the Uniontown, Pennsylvania, public schools and in 1949 received a B.S. degree in Business Administration from Waynesburg College.

Grace's husband is a family practitioner and practices in Woodbury. They have two children: Dr. Suzanne Holdcraft-Sherrare and David.

Mrs. Holdcraft is a charter member of the Gloucester County Visiting Homemaker Service. She sat on the Comprehensive Health Planning Board and its successor, Health Systems Agency, from 1970 to 1982 and served as an officer in the County Council and on the Executive Board, also serving as a member of numerous committees within the agency. Grace also is an active member of the Delaware Valley Alumni Association of Waynesburg College and has worked as a volunteer at Underwood Memorial Hospital, with the Girl Scouts, Boy Scouts, and the United Way.

Her MSNJ Auxiliary involvement includes many county chairmanships, and president of the Gloucester County Medical Society Auxiliary from 1965 to 1967.

At the state level, Mrs. Holdcraft served as state chairman of Health Careers, Safety and Disaster Preparedness, Legislation, Nominations, and Co-chairman of Convention. As president-elect, she conducted the state workshop and served as membership chairman.

Grace and her husband are active participants in all county Medical Society and Auxiliary events.

AMNJ Report

Arthur Krosnick, M.D., of Princeton, has been selected President of the Academy of Medicine for the 1986 to 1987 academic year. The Academy, which is the approved accrediting body for the Accreditation Council for Continuing Medical Education in New Jersey, is the teaching arm of the Medical Society of New Jersey.

Arthur Krosnick has spent his entire medical career in the pursuit of excellence in the practice of his chosen subspecialty, diabetology, and in education of patients, physicians, and other health professionals, as well as the public-at-large. It has been a labor of love, which also has included public health and clinical research.

Born and educated in Trenton. Dr. Krosnick was graduated from Temple University and Temple Medical School with an interval for U.S. Navy service aboard the USS Burleson as a landing craft officer in World War II from 1942 to 1945. After an internship at Mercer Hospital in Trenton, he completed residency training in internal medicine and diabetes at the University of Pennsylvania's Presbyterian Hospital and the Graduate Hospital in Philadelphia.

Dr. Krosnick has been Editor of the *Journal of the Medical Society of New Jersey*, now *NEW JERSEY MEDICINE*, since 1973 and has been a member of MSNJ for more than 30 years. He also is the Editor-in-Chief of *Diabetes Forecast*, the nation's foremost diabetes education publication for patients, published by the American Diabetes Association. His own publications include scientific articles, editorials, chapters, film scenarios, and teaching machine programs. He lectures on diabetes throughout the nation to physicians, nurses, pharmacists, dietitians, and patients.

Life has not been all medicine and science in the Krosnick home. His wife, Evelyn, manages the Greater Princeton Youth Orchestra. Their son, Jon, recently was awarded a Ph.D. degree at the University of Michigan's Institute for Social Research and now is an assistant professor in social psychology at Ohio State University. After graduating from Yale University with a bachelor's degree in biology and a master's degree in music, their daughter, Jody, entered Temple Medical School, where she is a sophomore with an eye on a career in plastic surgery.

This is the second honor bestowed on Arthur Krosnick by the Academy, which granted him its coveted Edward J. Ill Award in 1982. With pressures outside of medicine mounting to the bursting point, Arthur Krosnick's message to his colleagues inside and outside the Academy is: "Your expertise as a medical professional encompasses years of training, hundreds of case experiences, and continuing medical education. You are challenged daily by a demanding public, a carping government and its assignees, and a rapidly advancing front of technology and new scientific information. Your reasons for becoming a physician, the greatest calling for any man or woman, are not changed. People change. Society changes. But, their need for medicine, dentistry, and the allied sciences grows larger—not smaller. The pedestal on which American physicians have stood for so long is being chipped away, but the unparalleled degree of personal satisfaction in our service to humanity cannot be eroded by social forces. Now, indeed, is the time for us to come together."

Physicians Seeking Location in New Jersey

The following physicians have written to the Executive Offices of MSNJ seeking information on possible opportunities for practice in New Jersey. The information listed below has been supplied by the physician. If you are interested in any further information concerning these physicians, we suggest you make inquiries directly to them.

ANESTHESIOLOGY—Julio Araoz, M.D., 4 Avon Way, Parlin, NJ 08859. San Andres (Bolivia) 1959. Board eligible. Any type practice. Available.

Yellagondahalli V. Janardhan, M.D., 18 Agatha Dr., Edison, NJ 08817. Bangalore (India) 1971. Board certified. Group, partnership, fee for service. Available.

Ashokkumar J. Patel, M.D., 500 Central Ave., Apt. 1113, Union City, NJ 07087. M.P. Shah (India) 1977. Board eligible. Group, partnership, solo, hospital based. Available.

CARDIOLOGY—Mian Arshad Jan, M.D., 6901 Old York Rd., Apt. B-208, Philadelphia, PA 19126. Khyber Medical (Pakistan) 1974. Also, internal medicine. Board eligible. Board certified (IM). Group or partnership. Available July 1986.

EMERGENCY MEDICINE—Julian A. Sellek, M.D., 1501 S. Greenway, Coral Gables, FL 33134. Salamanca (Spain) 1963. Also, family practice. Board eligible. Board certified (FP). Group, solo, partnership. Available.

ENDOCRINOLOGY—Michael J. Miller, M.D., 2375 Hudson Ter., Apt. 3-E, Fort Lee, NJ 07024. SUNY-Downstate 1981. Also, internal medicine. Board eligible. Board certified (IM). Group or partnership. Available July 1986.

FAMILY MEDICINE—James Ditaranto, D.O., 3901 Manayunk Ave., Philadelphia, PA 19128. Osteopathic (Philadelphia) 1984. Board eligible. Group or partnership. Available.

Lawrence S. Gordon, M.D., 1016 Spinnaker Way, Apt. G-1, Melbourne, FL 32935. Rutgers 1982. Board certified. Group, partnership, solo. Available.

Natalia Moishezon, M.D., 207 Christie St., Leonia, NJ 07605. Moscow 1964. Board eligible. Group, partnership, industry. Available.

Jeffrey C. Schultz, M.D., 301 Victoria Dr., Monroeville, PA 15146. University of Massachusetts 1983. Board eligible. Group. Available July 1986.

GASTROENTEROLOGY—Richard J. Fastiggi, M.D., 10 Silver Stirrup Ct., Timonium, MD 21093. UMDNJ 1977. Board certified (IM). Solo, partnership, group. Available July 1986.

Prasad Satyanarayana Maturu, M.D., 8863 17th Ave., Brooklyn, NY 11214. Mysore Medical (India) 1977. Board eligible. Group or partnership. Available.

Brian C. Weiner, M.D., 24 Bowling Green Pl., Staten Island, NY 10314. SUNY-Downstate 1981. Board certified. Group or partnership. Available July 1986.

INTERNAL MEDICINE—Walter M. Bakun, M.D., 199 Casterline Rd., Denville, NJ 07834. St. George's University (Grenada) 1983. Board eligible. Group, partnership, solo. Available July 1986.

Eli R. Goldner, M.D., 2841 W. Rosemont, Chicago, IL 60659. Rome (Italy) 1980. Board eligible. Group, HMO, partnership. Available.

Arpad C. Heinrich, M.D., 360 W. Pleasantview Ave., Hackensack, NJ 07601. Temeschwar (Transylvania) 1980. Board eligible. Solo, partnership, group. Available.

Mian Arshad Jan, M.D., 6901 Old York Rd., Apt. B-208, Philadelphia, PA 19126. Khyber Medical (Pakistan) 1974. Also, cardiology. Board certified. Board eligible (CARD). Group or partnership. Available July 1986.

Gerald B. Miele, M.D., 293 Demott St., Mineola, NY 11501. Rush 1983. Board eligible. Solo or partnership. Available July 1986.

Michael J. Miller, M.D., 2375 Hudson Ter., Apt. 3-E, Fort Lee, NJ 07024. SUNY-Downstate 1981. Also endocrinology. Board certified. Board eligible (ENDOCRIN). Group or partnership. Available July 1986.

Vijay Mittal, M.D., 35-44 28th St., Apt. 5-C, Long Island City, NY 11106. All-India 1978. Board eligible. Group or solo (part-time). Available July 1986.

Makkena Ramachandra Rao, M.D., 501 Indian Creek Dr., Wilkes-Barre, PA 18702. Guntur Medical (India) 1974. Board eligible. Group or partnership. Available July 1986.

Danuta Silber, M.D., 340 West End Ave., NY 10023. UMDNJ (PGY-3), Poland 1979. Board eligible. Group or partnership. Available July 1986.

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Kirit Chandulal Shah, M.D., 50 S. Morrison, Apt. #7, Memphis, TN 38104. Grant Medical College (India) 1973. Board eligible. Board certified (IM). Partnership or group. Available September 1986.

Hal Tobias, M.D., 19456 NE 26th Ave., Apt. 14, North Miami Beach, FL 33180. Sackler (Israel) 1981. Board eligible. Group, partnership, solo. Available.

OBSTETRICS/GYNECOLOGY—Alan Cohen, M.D., 2335 Charney Rd., University Heights, OH 44118. Dominica 1982. Board eligible. Group, partnership, solo. Available July 1986.

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D. L. Johnson, M.D., 7216 Montrose Rd., Woodbury, MN 55125. Minnesota 1977. Board eligible. Group or partnership. Available.

PEDIATRICS—Ying-Jen Chen, M.D., 200-03 48th Ave., Bayside, NY 11364. Taipei Medical College (Taiwan) 1973. Board eligible. Partnership, solo, group. Available.

PULMONARY—Raymond Mantovani, M.D., 89 Fleet Pl., Mineola, NY 11501. Universidad Centro de Estudios Teenicos (Dominican Republic) 1981. Board eligible. Group, partnership, solo, hospital based. Available July 1986.

SURGERY, GENERAL—Bernard Henson, M.D., 1275 15th St., Apt. 16-0, Fort Lee, NJ 07024. Guadalajara (Mexico) 1979. Board eligible. Partnership or group. Available July 1986.

Patrick P. Montefusco, M.D., 4 Montfort Dr., Huntington, NY 11743. Georgetown 1977. Board certified. Partnership. Available.

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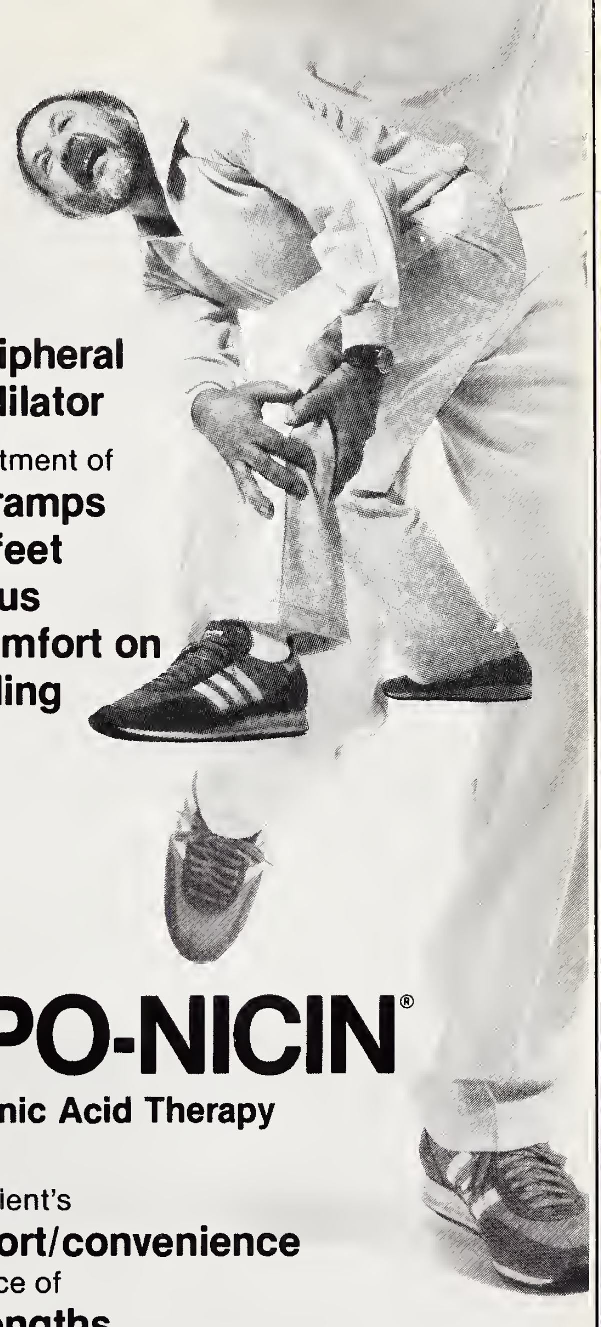

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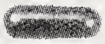
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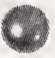
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CME CALENDAR

The following is a list of continuing medical education courses for the next two months. Contact the sponsoring organization for further information.

This list is compiled through the cooperation of the Committee on Medical Education of the Medical Society of New Jersey, The Academy of Medicine of New Jersey, the New Jersey Chapter of the American Academy of Family Physicians, and the Office of Continuing Medical Education of the UMDNJ. For information on accreditation, please contact the sponsoring organization(s), indicated by italics—last line of each item.

OBSTETRICS/GYNECOLOGY

July

Premenstrual Syndrome: What Do We Know? Where Do We Stand?

1 7:30-10 P.M.—Governor Morris Inn, Morristown

8 7:30-10 P.M.—The Clinton Inn, Tenafly

22 7:30-10 P.M.—Molly Pitcher Inn, Red Bank

29 7:30-10 P.M.—The Ramada Inn, Princeton
(Fair Oaks Hospital and AMNJ)

SURGERY

July

7 **Surgical Morbidity and Mortality**
14 **Conference**
21 4:30-6:30 P.M.—Medical Science
28 Bldg., B-610, NJ Medical School, Newark
(UMDNJ)

SURGICAL SPECIALTIES

July

2 **Common Orthopedic Problems at Knee and Shoulder**
1:30-2:30 P.M.—Rutgers Community Health Plan, 57 U.S. Hwy.1, New Brunswick
(UMDNJ and AMNJ)

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Contact the Academy of Medicine of New Jersey, Executive Offices for availability of these programs as well as registration fees.

Call (609) 896-1717.

LETTERS TO THE EDITOR

Certificate of Need; Research Physicians

Dear Dr. Krosnick:

The January 1986 issue of *NEW JERSEY MEDICINE* contained editorial comments about certificates of need and the possibility of procedural bias toward UMDNJ. I think an editorial response, clarifying the issues from the UMDNJ viewpoint, would be appropriate. This subject is no better exemplified than with magnetic resonance imaging (MRI). These facilities now are operating in New Jersey only in private offices, at great profit, despite the original intention of Commissioner Goldstein to furnish certificates of need for only four units in the state. The Middlesex-St. Peters unit in New Brunswick is at least six months from start-up. Similar considerations apply to the advent of ultrasonic lithotripsy in New Jersey. Currently active in a facility near Atlantic City, lithotripsy is a dream seemingly hopelessly tied up in certificate of need negotiations at Middlesex General-University Hospital.

I submit to you that the *NEW JERSEY MEDICINE* editorial carried an unfortunate bias. Please inform your readers of the alternative point of view.

(signed) Jeffrey L. Kaufman, M.D.
Assistant Professor of Surgery
UMDNJ-Rutgers Medical School

Dear Dr. Krosnick:

Dr. Kaufman's letter does not deal with the content nor tenor of my Hospital Governing Board article which appeared in the January 1986 issue of *NEW JERSEY MEDICINE.*

I maintained that the system has not worked, cannot work, and will not work. It is fatally flawed. It is subject to manipulation. It defies logic, economic, demographic, and sociologic reality. I always have maintained that application of the certificate of need process to a medical school seemed silly. If medical schools do not have ready access to state-of-the-art technology, they are not going to be able to produce state-of-the-art physicians.

The active lithotripsy unit in Marlton is not "near Atlantic City." It is affiliated with and sponsored by about eight hospitals which include those of UMDNJ at Cooper in Camden, and also the osteopathic school in Stratford. Consequently, I am not certain what his point is in raising that issue. I certainly acknowledge there is a quagmire. It is caused, however, by the very structure and operation of the certificate of need process—not the private practitioner.

I have no objection to UMDNJ presenting any or many editorials in *NEW JERSEY MEDICINE* on the certificate of need topic. I suspect that if they read my article carefully, they would support the conclusion that the certificate of need process is a mess, is not cost effective, and should be repealed. The federal department of Health and Human Services has filed a report with Congress calling for repeal of federal certificate of need requirements.

(signed) Vincent A. Maressa

Research Physicians

Dear Dr. Krosnick:

The need for clinical research in community hospitals and the doctor's office is a top priority in the United States, if this country is to stay competitive with Japan, Germany, and England as innovators of modern drugs. With DRG limiting hospital stays, and utilization preventing inhospital evaluation of antibiotics and other drugs, it is impossible to study new therapies for osteomyelitis and endocarditis today. To circumvent these cost-containing hospital measures, these diseases now are treated, for the most part, in the home. Therefore, previous investigators do not have access to such patients. Hence, studies will have to be done in a cooperative fashion with primary care doctors who are meticulous in following detailed protocols carefully.

Now, under the aegis of the Academy of Medicine of New Jersey, all physicians can apply to perform studies in their offices. Such physicians will be trained at Saint Michael's Medical Center and will work under an established investigator and protocol-liaison person. The doctors will receive an appropriate stipend for their time and effort for evaluable cases. This is an excellent opportunity to become involved in medicine on the horizon. Liability insurance is well covered by the pharmaceutical companies who also foster your presentations at national and international meetings.

If you are interested in participating, expanding your horizons, and think that you enjoy clinical research, send me your curriculum vitae with a summary of the type of patients you see, i.e. infections, heart, lung.

(signed) Leon G. Smith, M.D.
Director of Medicine
Saint Michael's Medical Center

BOOK REVIEWS

The Pancreas; Pediatric Neurosonography; Traveler's Medical Manual; Toxicology of the Eye, Ear, and Other Special Senses

The Pancreas: Principles of Medical and Surgical Practice

Luis H. Toledo-Pereyra (ed). New York, NY, John Wiley & Sons, Inc., 1985. Pp. 496. Illustrated. ($55)

This book was written in an attempt to review all medical and surgical aspects of pancreatic disease in a systematic fashion. As suggested in the foreword, it provides the clinician, regardless of his or her field of interest, with a review of the diagnosis and treatment of many problems involving the pancreas.

The publication date is listed as January 4, 1985. Therefore, the individual manuscripts would have been submitted in 1984. This may account for certain controversial topics being dealt with in a rather brief fashion or not at all.

For example, pancreas divisum is an anomaly that is being diagnosed with increasing frequency. The relationship between pancreas divisum and pancreatic disease has been debated in multiple reports. Although it is discussed, the sections are rather brief and, therefore, disappointing. Likewise, the use of pancreatic enzyme in the management of the pain of idiopathic chronic pancreatitis is not mentioned. The role of peritoneal dialysis in acute hemorrhagic pancreatitis has received increasing attention in the medical and gastroenterological literature, but is incompletely discussed here.

Despite these criticisms, I strongly endorse this textbook for any clinician studying or dealing with pancreatic disease. I believe it should be in all hospital libraries. As an internist and gastroenterologist, I particularly enjoyed and benefited from the surgical and pediatric sections. The section dealing with transplantation of a donor pancreas or islets was written clearly. This book presents the problems of the pancreas gland in a concise and interesting fashion and, for the most part, spells out current approaches to this management.

Joel P. Levinson, M.D.

Pediatric Neurosonography: Clinical, Tomographic and Neuropathologic Correlates

Fischer, Anderson, Shuman, and Stinson. New York, NY, John Wiley & Sons, 1985. Pp. 267. Illustrated. ($45)

This is an atlas with a succinct and clear text that one can read through at one sitting and then repeatedly return to with ever-increasing appreciation. The reflection of high-frequency sound from tissue forms the basis of sonographic imaging. Because the planes used in neurosonography are quite unlike CT scans or contrast x-rays, an atlas is a must as a ready reference format. Cranial sonography does not replace the CT, but is ideal for defining the central nervous system in a complementary fashion. This book integrates basic principles of ultrasound, covers technique, selection of equipment, and offers pictures and text for each pathological entity, its clinical correlates, and its variations from the normal anatomy of the developing infant. The juxtaposition of anatomic and/or pathological illustrations with pictures of real-time ultrasound studies makes this a valuable self-teaching tool. In its ready reference format, the book belongs on the shelf of pediatricians, neonatologists, neurologists, neurosurgeons, and radiologists, and in all nurseries.

With the survival of increasingly more immature infants, the incidence of intracerebral hemorrhage, malformations, trauma, or hydrocephalus rises. Neurosonography can be brought to the incubator and greatly reduces the need for x-ray. It offers a rapid, economical, readily reproducible testing device with which to diagnose and follow the troubled infant in the hospital or in the office. Because the premature brain evolves so rapidly, an atlas correlating change from 24 to 44 weeks' gestation is a must to differentiate lesions from the developing normal anatomy in the approaching-term infant.

You will refer to it frequently.

Henry R. Liss, M.D.

The Traveler's Medical Manual

Angelo T. Scotti, M.D. New York, NY, Berkley Books, 1985. Pp. 243. Paperback. ($4.95)

Traveling has become a major vacation and business experience for millions of Americans. They are alerted in many ways by travel agents on how to plan, anticipate, and prepare for their trips overseas; what to pack and what to avoid. They are cautioned "not to leave home without it," meaning their credit cards or travelers checks. It also would be prudent to prepare for unexpected medical emergencies that could occur on the trip.

Angelo T. Scotti, M.D., has been a director of the emergency room, Bayshore Community Hospital, Holmdel, for many years. He is well qualilfied by training and experience to handle any medical or surgical emergency, either on his own or by direction to the proper consultant. He has written *The Traveler's Medical Manual* and small, compact paperbacks that easily can be carried.

The Manual begins with a brief

checklist of simple medicinals and supplies one can carry along, then follows with summary descriptions of the more common medical emergencies and their resolutions. The thoughtful traveler might well be the good samaritan to care for the unexpected illness or accident of others in the group.

There is a worldwide directory of foreign countries that covers an A to Z listing, with the predominant risks and/or infectious diseases of the specific country. Reference is made, for almost every country, to the International Association for Medical Assistance to Travelers (IAMAT) with the name of the physician coordinator, the address, and telephone number to call, and the name of the hospital or clinic in each country.

For its price, size, and information, this book is a good investment towards a healthy, enjoyable vacation or business trip—so don't leave home without it.

Harry M. Poppick, M.D.

Toxicology of the Eye, Ear, and Other Special Senses

A. Wallace Hayes, Ph.D., (ed). New York, NY, Raven Press, 1985. Pp. 254. Illustrated. ($59.50)

This is an excellent small book; for the nonspecialist it reviews carefully the anatomy and physiology of the eye, ear, and olfactory system. Even though the specialist has had this information in his training, it still is a very good review. More important, the toxicology part of the book is something that is generally not heard in any residency program and, therefore, should be in the armamentarium of every physician.

The details of visual dysfunction and auditory dysfunction are covered thoroughly so that the practitioner readily can understand the effects on the eye and the ear from the toxic substances that are prevalent in our environment today. It is important to note that many of the so-called toxic substances are in the field of antibiotics and other medications that are used every day by physicians in their practice. There are some chapters which explain laboratory methods used in animals to understand more fully the toxic effects to various organs.

Therefore, I fully recommend this book for every physician in practice to cover a much-needed area where there is very little available in the literature and very few courses are given. This book is one of the few places where the needed information on toxic effects of our environment to the eye, ear, and other special senses can be found.

Leon G. Smith, M.D.

Now Available: 4th Edition, 1986

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OBITUARIES

Drs. Borrella; Della Fera; Gianni; Goracci; Hertzberg; Johnson; Malfara; McClintock; Mulholland; Murphy; Nunes; Pierson; Pollard; Sarla; Sena

Dr. Dominic D. Borrella

A general practitioner with an office in the Chambersburg area of Trenton for 50 years, Dominic D. Borrella, M.D., died at his home on January 17, 1986. Born in Philadelphia, Dr. Borrella was graduated from Georgetown University School of Medicine in 1935. He had been a member of our Mercer County component. During World War II, Dr. Borrella served as a flight surgeon in the Army Air Corps, in the China-India-Burma campaigns, and received many decorations for valor including the Asiatic-Pacific Battle Ribbon with three battle stars.

Dr. Lucien F. Della Fera

We just have learned of the death on November 22, 1985, of Lucien F. Della Fera, M.D., an ear, nose, and throat specialist with offices in Bloomfield. Born in 1907, in Newark, Dr. Della Fera received his medical degree from St. Louis University School of Medicine, Missouri, in 1935. He had been a member of our Essex County component, and of the American Medical Association. Dr. Della Fera was chief of the ENT department at Columbus Hospital, Newark, and had been affiliated with Lutheran Memorial Hospital, Newark, and St. Mary's Hospital, Passaic. During World War II, he was with the medical department of the United States Air Force, and held the rank of captain. In 1985, he was a recipient of MSNJ's Golden Merit Award, honoring his 50 years in medicine.

Dr. Angelo R. Gianni

A family practitioner in Netcong for many years, Angelo Rosario Gianni, M.D., died on February 15, 1986, at the age of 79. Born in New York City, Dr. Gianni was graduated from the Faculty of Medicine, University of Rome (Italy), in 1937. He had been a member of our Morris County component and of the American Medical Association. He had been affiliated with Dover General Hospital. During World War II, Dr. Gianni served with the medical corps of the United States Army, emerging with the rank of captain.

Dr. Armando F. Goracci

The 189th President of the Medical Society of New Jersey, Armando F. Goracci, M.D., died suddenly at his home, on February 7, 1986, at the untimely age of 69. Born in Old Forge, Pennsylvania, Dr. Goracci received his medical degree from the University of St. Louis, Missouri, in 1943, and upon his return to the area, became a member of the Philadelphia County Medical Society. In 1967, he joined the Gloucester County component of MSNJ, having established a surgical practice in Woodbury. Dr. Goracci was a Fellow of the American College of Surgeons and a Diplomate in surgery. He had been clinical assistant professor of surgery at Thomas Jefferson University Hospital, Philadelphia. Dr. Goracci was chief of surgery at Underwood Memorial Hospital from 1969 to 1976, and he had been on the surgical staff at Cooper Hospital-University Medical Center, Camden. His list of hospital affiliations during the length of his career was impressive, including: Philadelphia General Hospital; Doctor's Hospital, Philadelphia; Thomas Jefferson Hospital, Philadelphia; medical director and chief of surgery at Pennsylvania State Correctional Institution, Philadelphia. He had been active in county and state society affairs, including treasurer, secretary, and president of the Gloucester County Medical Society, president of the Medical Society of New Jersey in 1981 to 1982, a member of MSNJ's board of trustees from 1976. Dr. Goracci served on many Medical Society committees, among them the Councils on Legislation, Public Relations, and Public Health, as well as serving on the New Jersey Delegation to the AMA. During World War II, he served in the medical corps of the United States Army in Japan, with the rank of captain.

Dr. Irving Hertzberg

Irving Hertzberg, M.D., who practiced general medicine in Kearny for many years, died on August 15, 1985, at the age of 70. Born in New York City, Dr. Hertzberg earned his medical degree at Laval University Faculty of Medicine, Quebec, Canada, and was graduated in 1943. A member of our Essex County component and of the American Medical Association, Dr. Hertzberg had been affiliated with West Hudson Hospital, Kearny; Newark Beth Israel Medical Center; and the Clara Maass Memorial Hospital, Belleville. During World War II, he served in the medical corps of the Army of the United States, gaining the rank of major.

Dr. J. Randolph Johnson

James Randolph Johnson, M.D., an internist in Jersey City, died in University Hospital, Newark, on January 28, 1986, at only 59 years of age. A native of Jersey City, Dr. Johnson received his medical degree from New York University School of Medicine in 1951. He was a member of our Hudson County component, and of the American Medical Association. Dr. Johnson was board certified in his specialty, and a Fellow of the American College of Chest Physicians. He had been affiliated with the Medical Center and the B.S. Pollak Hospital, both in Jersey City. He had been a clinical instructor at Seton Hall College of Medicine, from 1958 to 1961, and at UMDNJ-New Jersey Medicial School, Newark, from 1962 to 1969. Dr. Johnson was involved in civic affairs: he had been a member of the NAACP, and was a member of the board of the Hudson County Action for Sickle Cell Anemia.

Dr. Joseph F. Malfara

After a long siege with cancer, Joseph Francis Malfara, M.D., died on January 29, 1986, at only 56 years of age. Born in Philadelphia, Dr. Malfara received his medical degree at Jefferson Medical College, Philadelphia, in 1956. He had been a member of our Camden County component, and of the American Medical Association. Dr. Malfara practiced family medicine, and had been affiliated with Garden State Community Hospital, Marlton.

Dr. Elsie McClintock

A physician involved in health education prior to her retirement, Elsie McClintock, M.D., died on September 23, 1985, at the venerable age of 86. Born in Rochester, New York, Dr. McClintock was awarded her medical degree in 1915 at Tufts University School of Medicine, Boston. She had been a member of our Union County component, and of the American Medical Association.

Dr. Robert E. Mulholland

A victim of heart failure, Robert E. Mulholland, M.D., died on January 24, 1986, at Dover General Hospital and Medical Center, at the untimely age of 62. Born in Council Bluffs, Iowa, Dr. Mulholland received his medical degree at New York Medical College in 1952. He maintained an office in Montville, had been chairman of the anesthesiology department at St. Clare's Hospital, Denville, and had been affiliated with All Souls Hospital, Morristown and Riverside Hospital, Boonton. Dr. Mulholland also served the transitional and cottage units at Greystone Park State Psychiatric Hospital, Parsippany. He had been a member of our Morris County component, and was widely known, along with his wife Mary, as founder of the Dope Open charity golf and tennis tournament, the proceeds from which were distributed to numerous drug and alcohol abuse centers in New Jersey.

Dr. George E. Murphy

George E. Murphy, M.D., a general surgeon who maintained a practice in Upper Montclair for many years before moving to Saddle River, died on September 11, 1985, at the age of 71. Born in New Jersey, Dr. Murphy was awarded his medical degree at Georgetown University School of Medicine, in 1940. Originally a member of our Passaic County component, he later became a member of our Essex County component and also of the American Medical Association. Dr. Murphy was a Fellow of the American Society of Abdominal Surgeons. He had been affiliated with St. Michael's Medical Center, Newark. During World War II, Dr. Murphy served in the medical department of the Army of the United States, attaining the rank of major.

Dr. Howard L. Nunes

A member of the Medical Society through our Hudson County component for over 40 years, Howard L. Nunes, M.D., died at Massachusetts General Hospital, Boston. Dr. Nunes was 67 years old at the time of his death. He had led a surgical-medical team of investigators at Seton Hall College of Medicine, West Orange, for many years. Dr. Nunes was a member of the American Medical Association, and was a Fellow of the American College of Surgeons.

Dr. J. Reginald Pierson

A retired Hopewell internist, Joseph Reginald Pierson, M.D., died on January 30, 1986, at The Medical Center at Princeton, at the advanced age of 83. A native of Hopewell, Dr. Pierson received his medical degree at the University of Pennsylvania School of Medicine, Philadelphia, in 1927. He was associated with his father in a practice from 1929 to 1958. Dr. Pierson was a member of our Mercer County component, and of the American Medical Association. He had been affiliated with Mercer Medical Center, Trenton, The Medical Center at Princeton, and St. Michael's Orphanage, Hopewell. During World War II, Dr. Pierson served in the medical department of the United States Army, with the rank of lieutenant-colonel.

Dr. William E. Pollard

A Princeton obstetrician and gynecologist, who retired in 1982, William Edward Pollard, M.D., died at his home, at the age of 79. A native of Lovington, Illinois, Dr. Pollard earned his medical degree at Columbia University College of Physicians and Surgeons in 1935. He had been a member of our Mercer County component, and for many years had served as chairman of the department of obstetrics and gynecology at The Medical Center at Princeton. He had been a consultant to Hunterdon Medical Center, Flemington, the Carrier Clinic, Belle Mead, and the New Jersey Psychiatric Institute, Princeton. Dr. Pollard was a Diplomate of the American Society of Obstetrics and Gynecology and a Fellow of the American College of Surgeons, and of the American College of Obstetrics and Gynecology. Dr. Pollard was known in the field of music; he had played cornet in the bands of Glen Miller and Benny Goodman.

Dr. Michael Sarla

A retired general practitioner who served for over 50 years in the Hackensack area, Michael Sarla, M.D., died on February 3, 1986, in Collegeville, Pennsylvania, at the remarkable age of 95. Born in Italy, Dr. Sarla emigrated to the United States, and obtained his medical degree from the Long Island College of Medicine (Brooklyn) in 1917. He had been a member of our Bergen County component and of the American Medical Association. Dr. Sarla had been affiliated with Hackensack Medical Center. In 1967, MSNJ conferred on Dr. Sarla its Golden Merit Award, honoring his 50 years as a physician.

Dr. Marie Coppola Sena

An administrative physician, who spent 28 years in civil defense and emergency medical services for the New Jersey State Department of Health, Marie Antoinette Coppola Sena, M.D., died on January 30, 1986, at Monmouth Medical Center, Long Branch. Dr. Sena, who retired 14 years ago, was 80 years old at the time of her death. Born in Stafford Springs, Connecticut, she was graduated from Women's Medical College, Philadelphia. Prior to her affiliation with the state of New Jersey, Dr. Sena had offices in New York City and Newark. She had been a member of our Essex County component, and of the American Medical Association. In 1980, Dr. Sena was a recipient of MSNJ's Golden Merit Award, honoring her 50 years in medicine.

AUTHOR INFORMATION

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CONTENT

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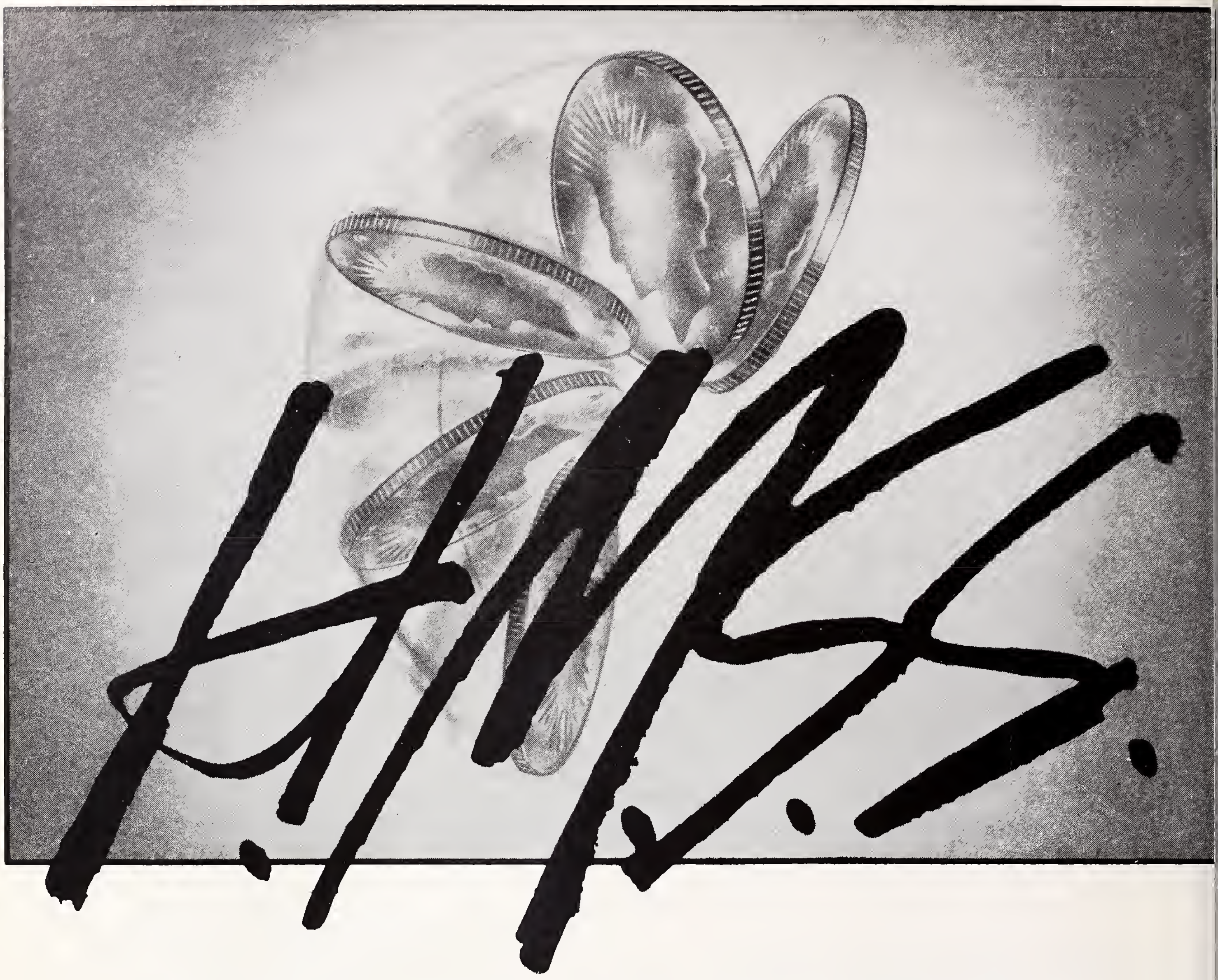

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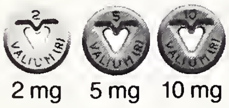

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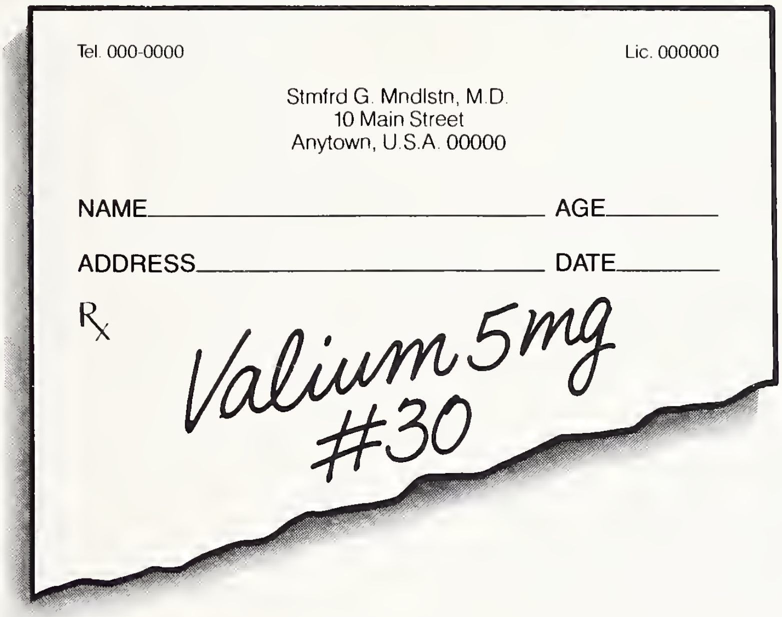

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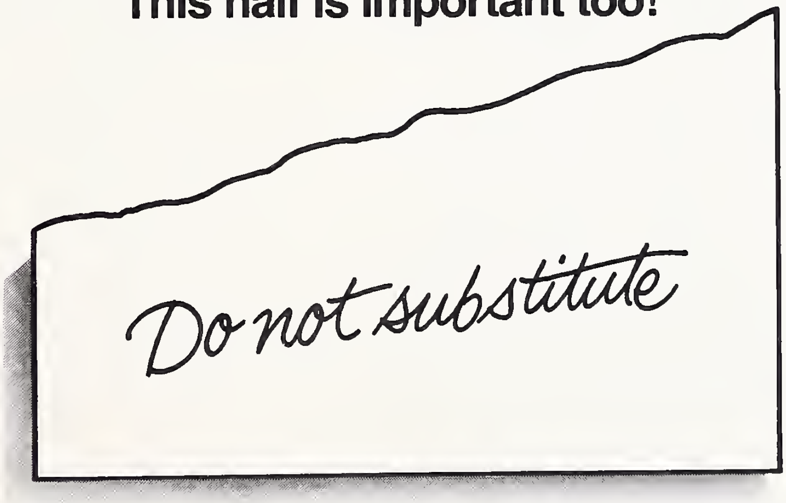

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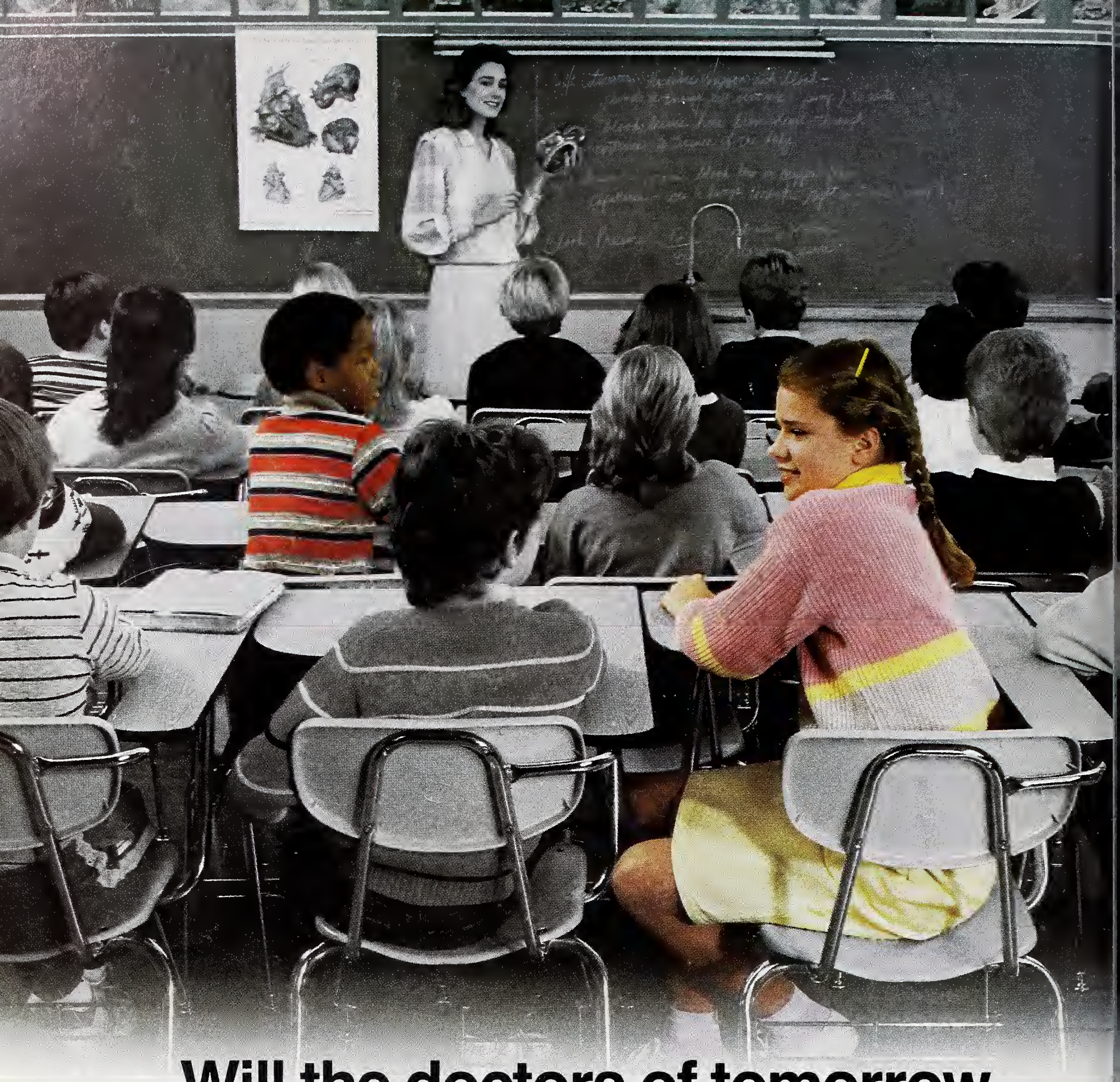

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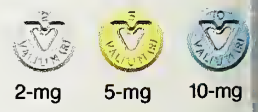

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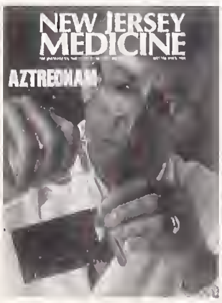
On The Cover: With this special issue, we present information on a new antibiotic called aztreonam. On the cover is Dr. Richard Sykes, working in his laboratory. Photograph by Charlotte Raymond.



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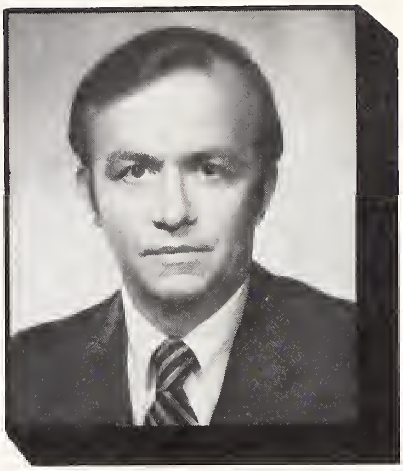
SPECIAL ISSUE: AZTREONAM

GERALD P. BODEY, M.D.



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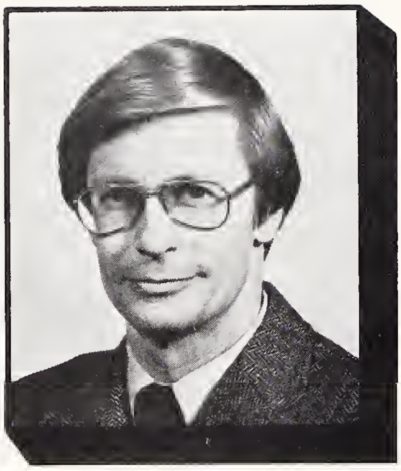

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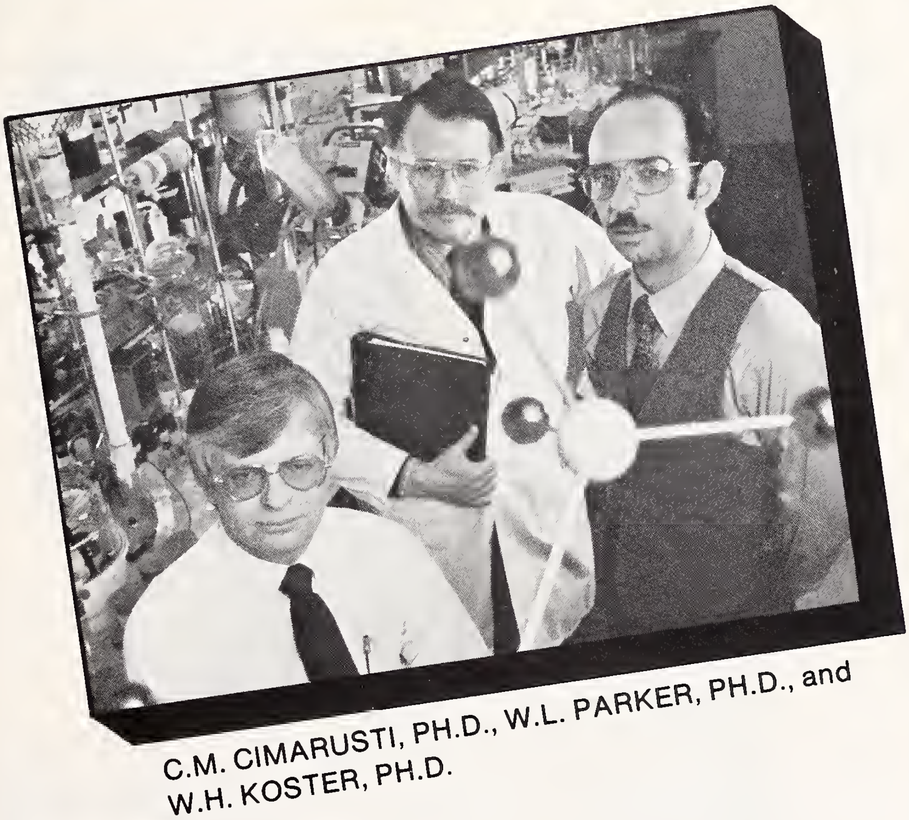

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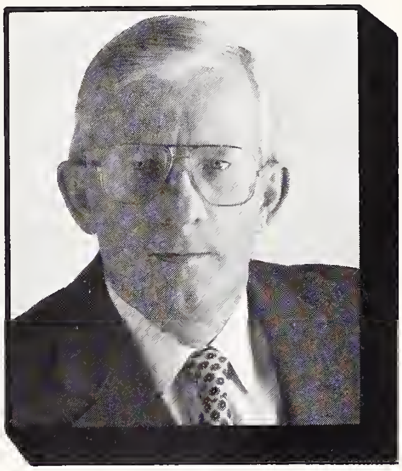


CECIL L. BENDUSH, M.D.

This special issue of *New Jersey Medicine* was developed to present our readers with information concerning a new antibiotic—Aztreonam.

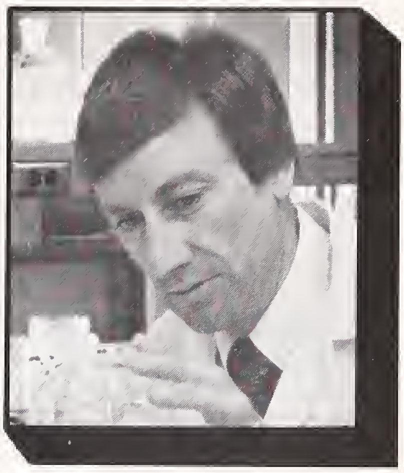
Aztreonam is a product of New Jersey: of its soil from the Pine Barrens and of its inhabitants under the direction of Dr. Richard Sykes. We hope this issue brings to the forefront the importance of this new antibiotic as well as the favorable prospects of other new drugs in the battle against infectious diseases.

The Medical Society of New Jersey reminds the readers that it is neither promoting nor advocating use of this product, and is not financially involved with Aztreonam.

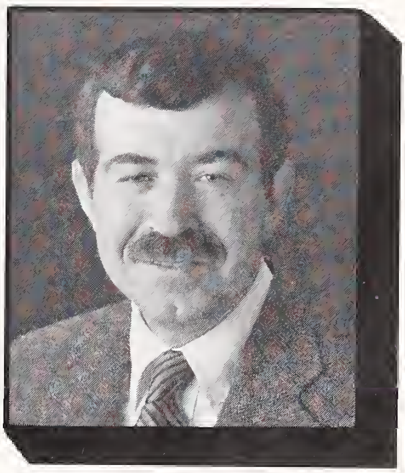
Leon G. Smith, M.D., Guest Editor
Arthur Krosnick, M.D., Editor
Geraldine R. Hutner, Managing Editor

SPECIAL ISSUE: AZTREONAM

LEON G. SMITH, M.D.


RICHARD B. SYKES, PH.D.

BRUCE H. MIGDALOF, PH.D.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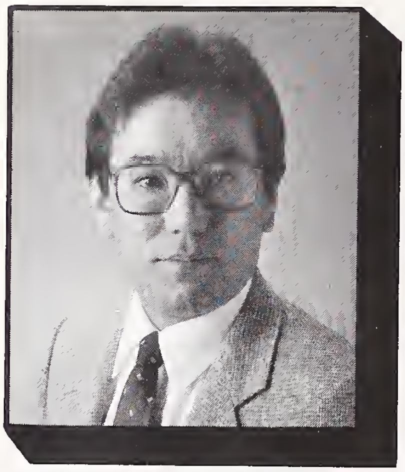
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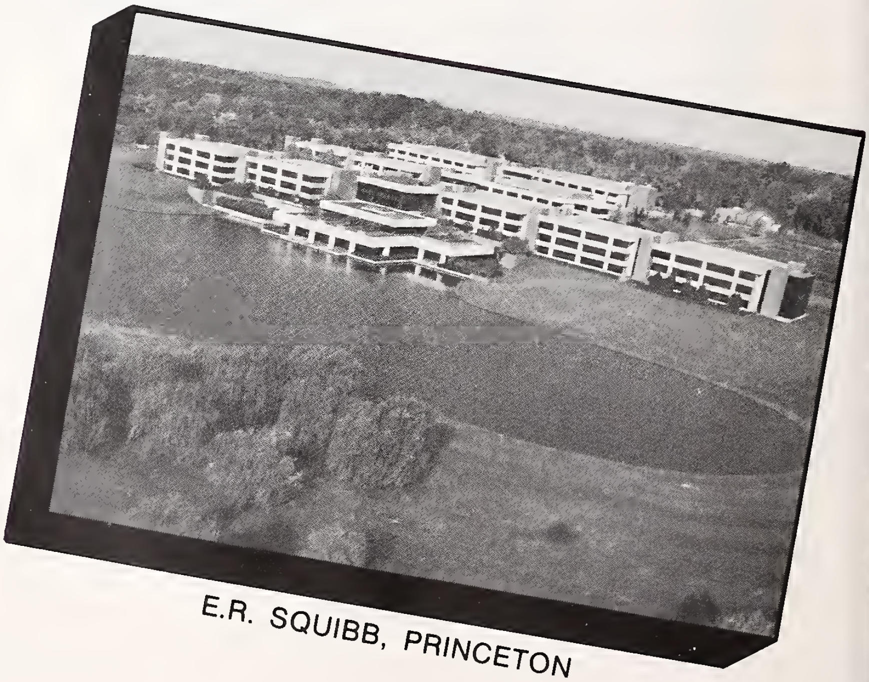
E.R. SQUIBB, PRINCETON

AZTREONAM: A MAJOR ANTIBIOTIC GIFT FROM NEW JERSEY TO MANKIND

LEON G. SMITH, M.D.

This special issue on aztreonam introduces the reader to a whole new family of β-lactam antibiotics called monobactams.

This special issue is dedicated to Dr. Richard B. Sykes and his colleagues from the Squibb Institute for Medical Research, Princeton, who have discovered a whole new family of β-lactam antibiotics called monobactams. The first monobactam was detected in a bacterium from the New Jersey Pine Barrens. Not since streptomycin was discovered by Waksman and his group at Rutgers, has New Jersey soil produced such a unique finding. Although Fleming discovered penicillin G in 1929, it was not until 1940 that Florey and Chain mass produced the first β-lactam (penicillin G) antibiotic. In 1945, Guiseppe Brotzu discovered the cephalosporins; the first general cephalothin was marketed by the Lilly Corporation in 1963.

Sykes et al. took the purple colony-forming *Chromobacterium violaceum* from the banks of the Wading River in the Pine Barrens in 1979. The antibiotic is unique for the following reasons:

1. There are no cross-sensitivity reactions with the penicillins.
2. It has excellent activity against gram-negative aerobic bacteria including *Pseudomonas aeruginosa.*
3. There is no pseudomembranous colitis.
4. There are no bleeding problems.
5. It can be totally synthesized in the laboratory.
6. It binds exclusively to PBP-3.

The talented Dr. Sykes discusses the historical development of aztreonam in a very concise British manner. Harold Neu, M.D., reviews his excellent results in urinary, respiratory, abdominal, gynecological, and bone and joint infections; he also compares aztreonam in vitro activity to other β-lactam antibiotics and demonstrates the narrow pure anti-gram negative spectrum of this new compound. The biological fate of aztreonam is compared to other β-lactam antibiotics by Bruce H. Migdalof, Ph.D., which includes placenta transfer, milk excretion, and disposition in tissue data. The St. Michael's Medical Center article by Edward Johnson, M.D., et al. demonstrates the safety of aztreonam in various clinical situations. Gerald Bodey, M.D., of the University of Texas, M.D. Anderson Hospital and Tumor Institute at Houston provides further data of the efficacy of this compound in seriously ill cancer patients with neutropenia. The other articles by Drs. Bendush and Bonner center on the clinical trials of aztreonam.

In conclusion, the soils of New Jersey once again have produced a new family of antibiotics. Aztreonam is a welcome new addition to the fight against serious infections of the gram-negative aerobic bacteria. It is safe and shall be used with great frequency throughout the world. Most importantly, aztreonam is the first of a whole new family of single, effective, safe antibiotics.

The Pine Barrens of South Jersey: A Discovery

Arthur Krosnick, M.D.*

In the 1800s, the hidden treasures of the Pine Barrens were used by "Doctor" James Still. Today, these treasures provided the basis for a new antibiotic developed by Dr. Richard Sykes.

Aztreonam, the principal character in this exposé, is a product of one of nature's wondrous secrets carefully hidden and hitherto undiscovered in New Jersey's vast and now-famous Pine Barrens. As Dr. Richard B. Sykes (page 8) points out, a "completely new technology" was implemented by collecting and examining soil and water samples from all over the world. To the good of mankind, careful chemical, pharmacological, and bacteriological research on samples from the area led to *Chromobacterium violaceum* and the monobactams.

But, Dr. Sykes and the E.R. Squibb scientists were not the first to make use of the hidden treasures of that legendary region of our remarkable state. The Pine Barrens, which stretch through most of the lower part of Burlington County and into Ocean County, contain two million acres of timberland and most of the remaining wildlife of South Jersey. The Pinelands and Pine Barrens of Cumberland County largely are occupied by the Wharton Tract.

The area has a unqiue history:

> The still undeveloped region was a haven for the Tories of Philadelphia and upper Jersey fleeing the wrath of the Continentals following the Revolution. It also was the jungle into which roving bandit bands such as the Mulliners and the Gibersons could disappear when law forces were too close on their trail. Early iron empire builders found that the Pine Barrens were lands of plenty with unlimited resources for their enterprises. Wandering preachers found the Barrens a place where their gospels of hellfire and brimstone were easily accepted. To Joseph Wharton, Philadelphia industrialist, the Barrens represented a vast watershed whose underground rivers would assure Philadelphia a pure water supply for generations. Others found the Barrens offered a way of life where few questions were asked and the pace was easy.[1]

One of the most interesting characters of the Pinelands was "Doctor" James Still, born of freed slaves, Levin and Charity Still, of Indian Mills in 1812. His only education was "three months' instruction in reading, writing, and numbering." Still never was a licensed physician nor a member of the Medical Society of New Jersey. An inveterate reader, Still "became fired with an ambition that was to dominate his life—to become a doctor—when he observed a Dr. Fort vaccinate the children of the area. Unable to obtain a formal medical education or apprenticeship, "James Still learned how to use herbs from his Indian neighbors, read medical books, and cared for the sick in the Pine Barrens in the mid 1800s."

Still's understanding of Indian practical know-how

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*Dr. Kosnick is the Editor of *NEW JERSEY MEDICINE*, the journal of the Medical Society of New Jersey.

and self-taught book knowledge stimulated him to buy distilling equipment and to make medicinals from "herbs furnished by the pines, such as sassafras root, skunk cabbage, and snakeroot." He served the Pineys far and wide by delivering medicine by horse and wagon. Still's reputation grew along the Pines and even spread to Charles and William Ellis, Philadelphia pharmacists, who "offered to buy all the mixtures he could give them." It was not long before "lines of horses and carriages clogged the road to the Still house" as people came to obtain his medicines.

Pharmaceutical remedies have become available over the eons by empirical means, i.e. by depending upon experience or observation alone without using science or theory. Modern ethical pharmaceutical corporations are not above checking out grandmother's nostrums in hopes of finding a clue. Another important source is serendipity, the faculty for making desirable discoveries by accident. Of course, this technique requires the knowledge and ability to recognize and utilize such discoveries. Finally, the diligent search for specific agents aimed at definite problems by application of the scientific method is the current method of procedure.

What do the "black doctor" of the Pine Barrens and the E.R. Squibb scientists have in common? They both scratched around the stunted pines and vegetation of the area, while the deer, fox, rabbits, quail, beavers, raccoons, and skunks, the original Pineys, stood aside and watched. They both hoped to find something of value to mankind and one must assume that they both succeeded.

What additional remedies are to be found in the Pine Barrens? Only God knows, but for now we can say that the discovery of the monobactams, which ultimately led to the synthesis and production of aztreonam, will add another notch to the considerable reputations of that extraordinary bit of relatively untouched New Jersey soil and of E.R. Squibb & Sons, Inc., a world leader in pharmaceutical research and production.

REFERENCE

1. McMahon W: *South Jersey Towns*. New Brunswick, NJ, Rutgers University Press, 1973.

AZTREONAM: DISCOVERY AND DEVELOPMENT OF THE MONOBACTAMS

RICHARD B. SYKES, PH.D., J.S. WELLS, PH.D., W.L. PARKER, PH.D.,
W.H. KOSTER, PH.D., C.M. CIMARUSTI, PH.D.*

Utilizing a specific screening technology to detect β-lactam antibiotics in nature, we discovered a new family of antibiotics, the monobactams. The first monobactam was detected in bacterium isolated from the New Jersey Pine Barrens.

Penicillin has remained a household term for almost 40 years. Since the introduction of the first naturally occurring penicillin (penicillin G) in the early 1940s, numerous semisynthetic penicillins have made their way to the marketplace as the need for newer and more potent antibiotics developed. Within the last 20 years, cephalosporins have made important contributions to the treatment of infectious diseases. Penicillins and cephalosporins are referred to as β-lactam antibiotics, a term reflected in the chemical structure of the four-membered nitrogen-containing ring common to these molecules (Figure 4).

PENICILLIN AND CEPHALOSPORIN

The discovery and development of penicillin has been retold many times, incorporating numerous personal and scientific perspectives. However, throughout the various interpretations one fact remains undisputed, the discovery of penicillin was a chance observation.

In 1945, Guiseppe Brotzu designed his experiments to look specifically for antibiotic activity among the organisms of sewage effluent at Su Siccu Beach, Cagliari, Sardinia. The activity pursued by Brotzu was that elaborated by a strain of *Cephalosporium acremonium* and subsequently identified as penicillin N.[1]

An additional antibiotic produced by Brotzu's fungus was discovered by workers at Oxford, England, only as an afterthought through careful experimentation and observation. This molecule named cephalosporin C is the progenitor of the cephalosporin family of antibiotics.[2]

For 15 years following the discovery of cephalosporin C, no additional naturally occurring β-lactam-containing molecules were reported. This observation becomes more remarkable when considered in relation to the fact that the period between 1955 to 1965 often is referred to as the "golden era of antibiotic discovery." By the 1970s, this situation changed dramatically with new and novel molecules being reported in relative abundance. This sudden change in fortune can be attributed to a number of factors foremost among which were the development of "mode of action screens" and significant advances in isolation technology.

NATURALLY OCCURRING β-LACTAMS

In 1971, Nagarajan and his colleagues reported their discovery of the cephamycins (7 α-methoxycephalosporins) as products of streptomycetes.[3] These molecules were detected in screens developed to look for agents producing morphological changes in sensitive

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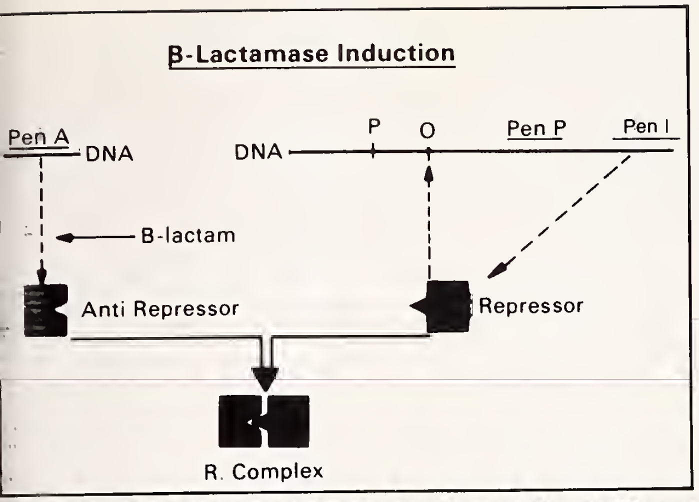


Figure 1—Model proposed for the regulation of penicillinase production. P and O are the promoter and operator sequences for the penicillinase structural gene (penP). PenI is a regulatory gene specifying the penicillinase repressor, and PenA is a regulatory region specifying a penicillinase antirepressor protein. Formation of the repressor/antirepressor complex (R complex) which takes place only when inducer molecules are present, inactivates the penicillinase repressor. Subsequently transcription occurs and penicillinase synthesis begins.

bacteria and thus represent the first naturally occurring β-lactams to emerge from directed screening procedures. Following the discovery of the cephamycins came clavulanic acid and the carbapenems, molecules detected on the basis of their ability to inhibit β-lactamases.[4]

One of the most successful directed screening technologies for β-lactams has been the use of β-lactam antibiotic-supersensitive mutants. Using a supersensitive mutant of *Pseudomonas aeruginosa*, Kitano and his colleagues reported on the production of penicillins and cephalosporins from fungal strains previously not associated with the production of these compounds.[5] In a similar vein, workers at Fujisawa discovered a new series of monocyclic β-lactams (nocardicins) produced by *Nocardia* sp., utilizing a supersensitive mutant of *Escherichia coli*.[6]

The latest development in this ever-broadening field has been the discovery of β-lactam antibiotics produced by bacteria. Within the last few years, bacteria have been shown to produce a series of novel monocyclic β-lactam antibiotics (monobactams), a carbapenem, and a series of cephalosporins.[7-10]

THE DISCOVERY OF AZTREONAM

The decision to search for novel naturally occurring β-lactam antibiotics at Squibb came in the late 1970s. The first step in this process was the design of a sensitive screening technology highly specific for β-lactam-containing molecules. This was achieved by developing screens to look for the induction of β-lactamases in bacteria.[11] Activated β-lactam-containing molecules interact with two main groups of enzymes: cell wall cross-linking enzymes, e.g. transpeptidases, and the β-lactam-hydrolyzing enzymes or β-lactamases.[12] At the inception of our studies the only substrates for β-lactamases were β-lactam antibiotics. In addition to acting as substrates, these β-lactam-containing molecules also possess the ability to induce β-lactamase production in certain strains of gram-positive and gram-negative bacteria. A model for regu-

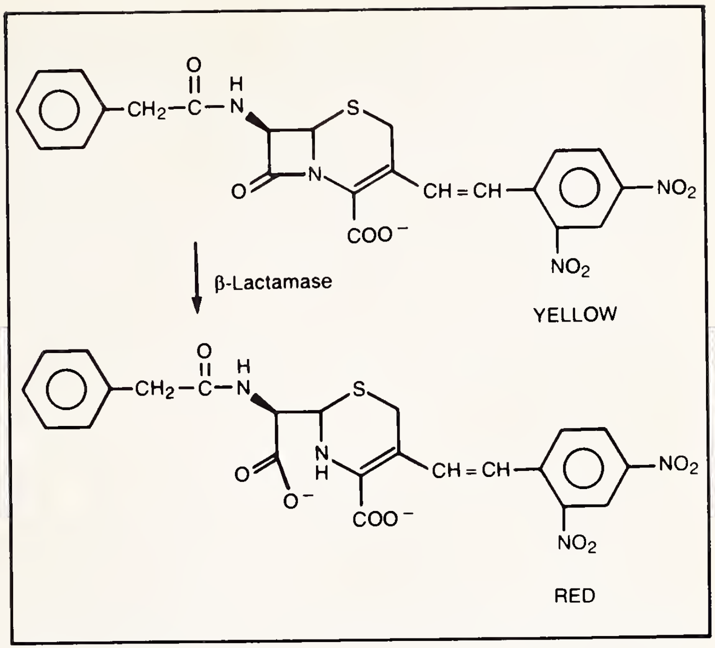


Figure 2—Enzymatic hydrolysis of the chromogenic cephalosporin SQ 24,962.

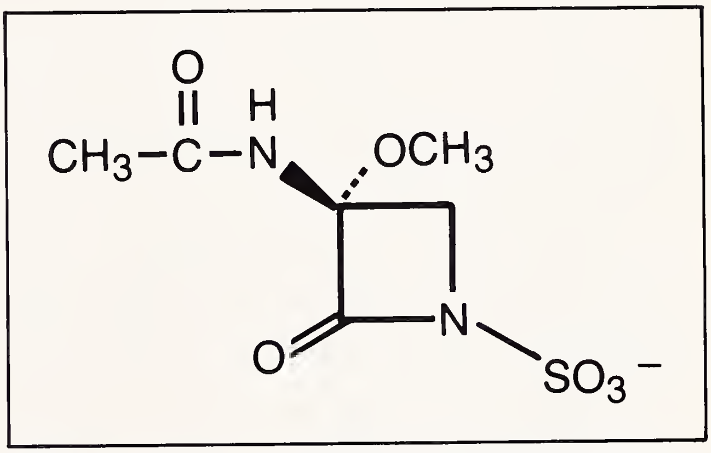


Figure 3—EM5117 (SQ 26,180) the first monobactam, produced by strains of *Chromobacterium violaceum*.

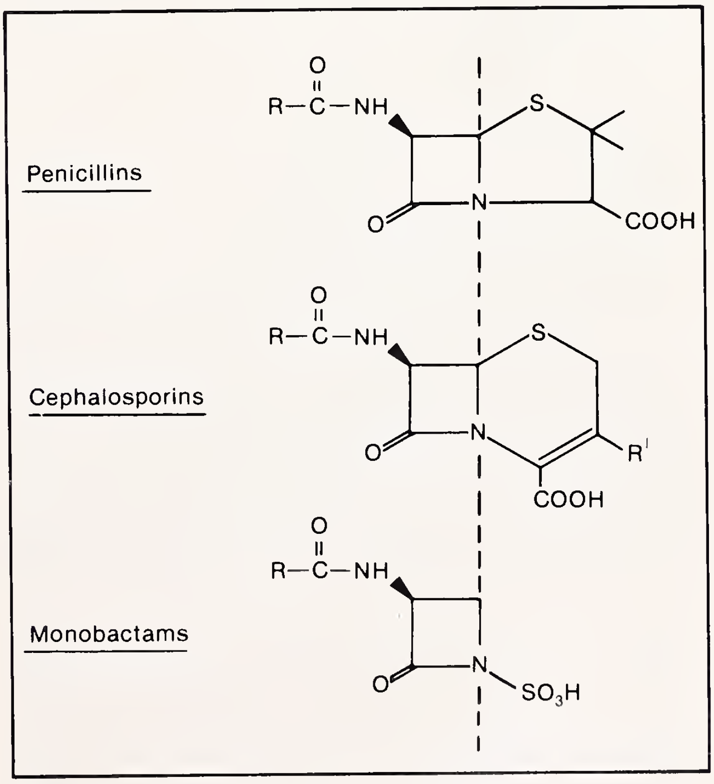
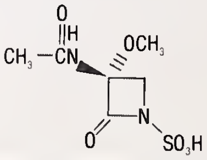
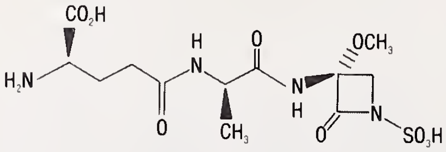
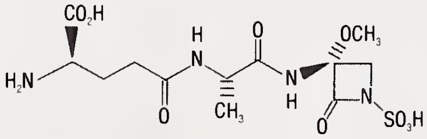
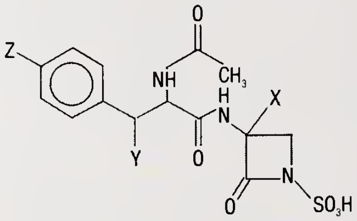
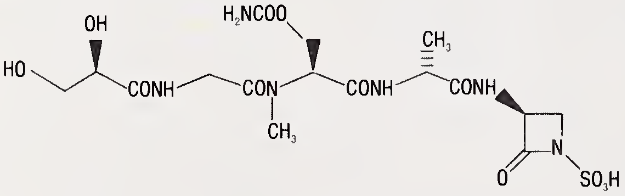
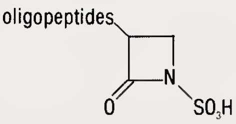


Figure 4—Comparative β-lactam structures.

TABLE 1

Structure and Source of Naturally Occurring Monobactams

Compounds	Structure	Producing Organism	Culture Reference	Reference
SQ 26,180		*Chromobacterium violaceum*	SC 11,378 (ATCC 31,532)	[16] [17] [18]
SQ 26,445 (sulfazecin)		*Gluconobacter* sp.	SC 11,435 (ATCC 31,581)	[16]
		Acetobacter pasteurianus subsp. *pasteurianus*	ATCC 6033	[19]
		Acetobacter aceti subsp. *aceti*	ATCC 15973	
		Acetobacter peroxydans	ATCC 12874	
		Acetobacter aceti subsp. *liquefaciens*	ATCC 23751	
		Acetobacter sp.	ATCC 21760	
		Gluconobacter oxydans subsp. *oxydans*	ATCC 19357 ATCC 15178	
		Gluconobacter oxydans subsp. *suboxydans*	ATCC 19441 ATCC 23773	
		Gluconobacter oxydans subsp. *industrialis*	ATCC 11894	
Isosulfazecin		*Pseudomonas mesoacidophila*	SB-72310	[20]
EM 5400		*Agrobacterium radiobacter*	SC 11,742	[16] [21] [22]
SQ 28,332		*Flexibacter* sp.	SC 11401 (ATCC 35,208)	[8]
SQ 28,502 SQ 28,503		*Flexibacter* sp.	ATCC 35103	[10]

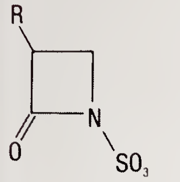
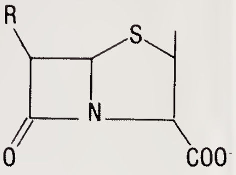
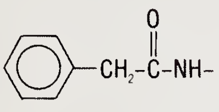
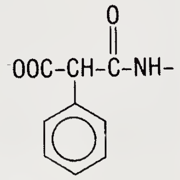
	X	Y	Z
SQ 26,823	OCH_3	H	H
SQ 26,875	OCH_3	H	OH
SQ 26,700	H	H	OH
SQ 26,970	OCH_3	OH	OSO_3H
SQ 26,812	OCH_3	OSO_3H	OSO_3H

lation of penicillinase production in gram-positive bacteria was proposed by Imsande, the main features of which are shown in Figure 1.[13] Having induced the enzyme, the next step is the use of a simple procedure to detect its presence. Over the years, many methods have been described for β-lactamase detection, one of the simplest being the use of chromogenic substrates.[14]

In one of our screens we employed a strain of *Bacillus licheniformis* that in the absence of β-lactam antibiotics produces minimal levels of β-lactamase activity. However, in the presence of β-lactams, large

TABLE 2

Antibacterial Relationships of Penicillins to Their Homologous Monobactams
Minimum Inhibitory Concentrations (μg/ml): Agar Dilution Assays*

 *Monobactam*  *Penicillin*			R								
			Nucleus	**Penicillin**		**Monobactam**		**Penicillin**		**Monobactam**	
			Test Compounds	**Benzyl Penicillin**		**SQ 26,324**		**Carbenicillin**		**SQ 81,324**	
			CFU	**10^4**		**10^4**		**10^4**		**10^4**	
Organism		**SC Number**									
Staphylococcus aureus	PENase−	2,399		<0.05		3.1		0.2		100	
Staphylococcus aureus	PENase+	2,400		3.1		6.3		6.3		100	
Escherichia coli		8,294		50		25		25		>100	
Klebsiella aerogenes		10,440		25		50		25		>100	
Proteus rettgeri		8,479		3.1		25		0.4		>100	
Enterobacter cloacae		8,236		>100		>100		12.5		100	
Pseudomonas aeruginosa		8,329		>100		>100		50		50	
			CFU	**10^4**	**10^6**	**10^4**	**10^6**	**10^4**	**10^6**	**10^4**	**10^6**
Escherichia coli	TEM+	10,404		>100	>100	50	>100	>100	>100	>100	>100
Escherichia coli	TEM−	10,439		12.5	25	12.5	25	6.3	12.5	>100	>100
Enterobacter cloacae	P99+	10,435		>100	>100	100	>100	>100	>100	>100	>100
Enterobacter cloacae	P99−	10,441		25	100	>100	>100	6.3	12.5	>100	>100
Klebsiella aerogenes	K1+	10,436		>100	>100	>100	>100	>100	>100	>100	>100
Klebsiella aerogenes	K1−	10,440		12.5	12.5	50	100	6.3	12.5	>100	>100

*Minimum inhibitory concentrations were determined by a twofold agar dilution method on DST agar.

amounts of enzyme are produced which can be detected in liquid or solid media by the use of the chromogenic cephalosporin substrate (Figure 2). This methodology has proved to be highly specific for β-lactam-containing molecules. The only false positives to emerge so far are a group of bacterially produced β-lactones.[15]

Having developed the screening system, the next step was its use in the screening of fermentation broths. Micro-organisms (streptomycetes and fungi), obtained from soil samples taken from various locations around the globe validated the test but failed to lead to the discovery of novel compounds. It was at this juncture in our program that a decision was made to examine soil isolates of bacteria. The first samples to be examined were taken from the banks of the Wading River in the Pine Barrens of New Jersey. These samples contained a number of purple colony-forming bacteria later identified as *Chromobacterium violaceum*.[16] Fermentation samples from these bacteria when subjected to the β-lactamase induction test indicated a positive response. This material designated EM5117 was later identified as a monocyclic β-lactam antibiotic (Figure 3).[17] Although strains of *C. violaceum* are relatively common in samples of soil and water, β-lactam producing strains appear to be restricted almost entirely to the area of the Pine Barrens.[15] We subsequent-

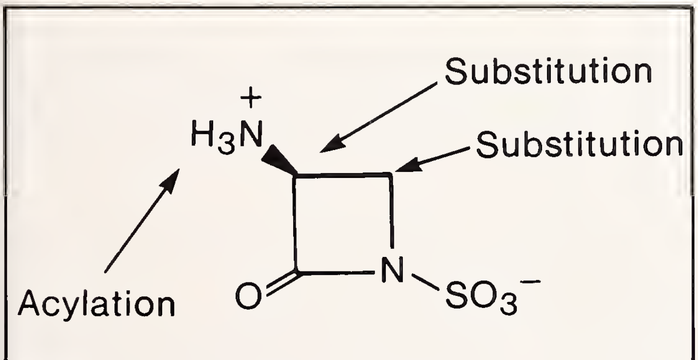


Figure 5—3-aminomonobactamic acid.

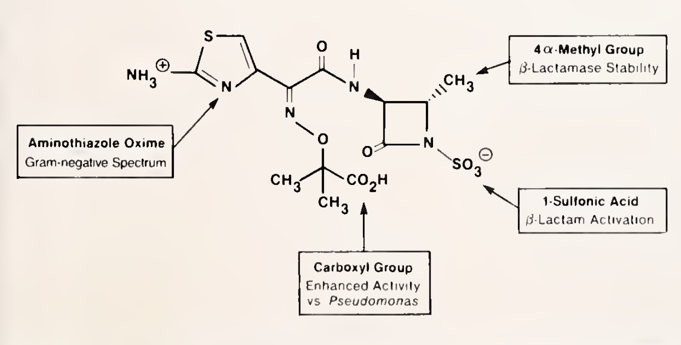
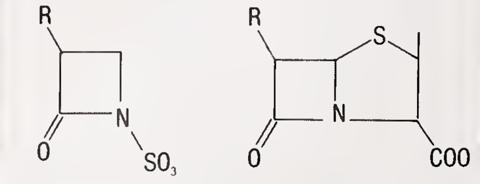
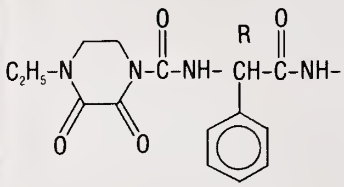
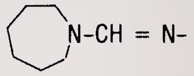


Figure 6—Structure-activity relationships in the aztreonam molecule.

TABLE 2

Antibacterial Relationships of Penicillins to Their Homologous Monobactams
Minimum Inhibitory Concentrations (μg/ml): Agar Dilution Assays*

 *Monobactam* *Penicillin*			**R**								
			Nucleus Test Compounds	**Penicillin**		**Monobactam**		**Penicillin**		**Monobactam**	
				Piperacillin		**SQ 81,427**		**Mecillinam**		**SQ 81,364**	
			CFU	**10^4**		**10^4**		**10^4**		**10^4**	
Organism		**SC Number**									
Staphylococcus aureus	PENase-	2,399		0.4		1.6		25		>100	
Staphylococcus aureus	PENase+	2,400		3.1		1.6		>100		>100	
Escherichia coli		8,294		3.1		0.8		<0.05		12.5	
Klebsiella aerogenes		10,440		12.5		1.6		<0.05		25	
Proteus rettgeri		8,479		0.1		0.1		0.1		12.5	
Enterobacter cloacae		8,236		1.6		0.8		0.1		25	
Pseudomonas aeruginosa		8,329		3.1		3.1		>100		>100	
			CFU	**10^4**	**10^6**	**10^4**	**10^6**	**10^4**	**10^6**	**10^4**	**10^6**
Escherichia coli	TEM+	10,404		>100	>100	25	>100	50	>100	>100	>100
Escherichia coli	TEM-	10,439		1.6	3.1	1.6	6.3	0.2	0.4	50	>100
Enterobacter cloacae	P99+	10,435		>100	>100	>100	>100	0.8	3.1	>100	>100
Enterobacter cloacae	P99-	10,441		1.6	3.1	0.4	1.6	0.2	0.4	50	>100
Klebsiella aerogenes	K1+	10,436		>100	>100	>100	>100	>100	>100	>100	>100
Klebsiella aerogenes	K1-	10,440		1.6	3.1	0.8	3.1	3.1	100	>100	>100

*Minimum inhibitory concentrations were determined by a twofold agar dilution method on DST agar.

ly have discovered a whole series of monocyclic β-lactam molecules produced by bacteria and given them the class name of monobactams. Naturally occurring monobactams reported to date are listed in Table 1.

The naturally occurring monobactams are characterized by the 2-oxoazetidine-1-sulfonic acid moiety having an acyl side chain in the 3β-position and in the majority of cases, a 3α-methoxy group (Table 1). The simple monocyclic structure of the monobactams starkly contrasts with the chemically complex bicyclic penicillin and cephalosporin molecules (Figure 4). Unlike the fermentation-derived penicillins and cephalosporins, the monobactams from nature exhibit poor antibacterial properties and as such cannot be considered useful antibiotics in their own right.[23]

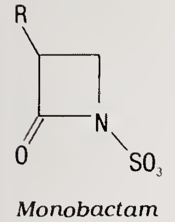
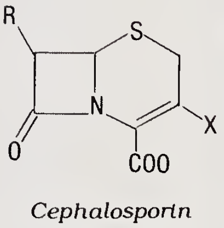
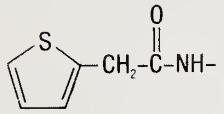
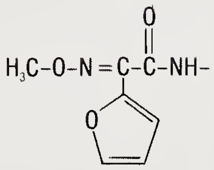
From previous experience of side chain modification with penicillins and cephalosporins, it was known that molecular modification on the C-3 amide residue could have a dramatic impact on the properties of the molecule. Alterations in the spectrum of activity among the naturally occurring side chain derivatives suggested this also would be true for the monobactams. In addition, the simple monobactam structure provided a template for molecular modifications aimed at increasing the activation of the β-lactam ring and providing adequate binding of the molecule to the enzyme's active site. Utilizing the synthetic monobactam nucleus, 3-aminomonobactamic acid (Figure 5), a large number of synthetic monobactam derivatives were made and tested for their biological properties.[24-26]

Initially, a series of molecules was synthesized bearing side chains gleaned from the most successful of the penicillins and cephalosoprins.

The same problems encountered in translating the activity of a penicillin to a cephalosporin also are met when attempting extrapolation to the activity of monobactams. In general, the monobactams are like the classical β-lactams whereby side chain substitution influences the spectrum of activity. Compounds may exhibit activity against gram-negative bacteria, gram-positive bacteria, or be broad spectrum in character. However, in terms of extremes of spectrum that can be achieved, the monobactams probably surpass their classical counterparts. Some comparisons to analogous penicillins are shown in Table 2. The penicillin G monobactam analog exhibits an antigram-positive spectrum but it is less active than penicillin G. Monobactams analogous to carbenicillin and mecillinam also are less active than their penicillin counterparts while the piperacillin monobactam is equal both in breadth of spectrum and level of activity to piperacillin.

TABLE 3

Antibacterial Relationships of Cephalosporins to Their Homologous Monobactams
Minimum Inhibitory Concentrations (μg/ml): Agar Dilution Assays*

 *Monobactam*  *Cephalosporin*			**R**								
			Nucleus Test Compounds	**Cephalosporin**		**Monobactam**		**Cephalosporin**		**Monobactam**	
				Cephalothin		**SQ 81,387**		**Cefuroxime**		**SQ 81,532**	
			CFU	**10^4**		**10^4**		**10^4**		**10^4**	
Organism		**SC Number**									
Staphylococcus aureus	PENase-	2,399		0.1		3.1		0.8		3.1	
Staphylococcus aureus	PENase+	2,400		0.2		3.1		0.8		6.3	
Escherichia coli		8,294		12.5		25		6.3		25	
Klebsiella aerogenes		10,440		6.3		50		6.3		50	
Proteus rettgeri		8,479		0.1		12.5		>0.05		12.5	
Enterobacter cloacae		8,236		>100		100		6.3		12.5	
Pseudomonas aeruginosa		8,329		>100		>100		>100		100	
			CFU	**10^4**	**10^6**	**10^4**	**10^6**	**10^4**	**10^6**	**10^4**	**10^6**
Escherichia coli	TEM+	10,404		6.3	>100	50	>100	3.1	6.3	>100	>100
Escherichia coli	TEM-	10,439		1.6	25	50	50	3.1	6.3	25	25
Enterobacter cloacae	P99+	10,435		>100	>100	>100	>100	>100	>100	>100	>100
Enterobacter cloacae	P99-	10,441		6.3	12.5	25	50	25	12.5	50	50
Klebsiella aerogenes	K1+	10,436		>100	>100	>100	>100	>100	>100	>100	>100
Klebsiella aerogenes	K1-	10,440		3.1	3.1	100	100	1.6	3.1	25	50

*Minimum inhibitory concentrations were determined by a twofold agar dilution method on DST agar.

A similar situation is seen in Table 3 where monobactams are compared to their cephalosporin counterparts. The cephalothin analog reflects the spectrum of activity but is less potent than cephalothin. The cefoperazone analog on the other hand, closely reflects the activity of cefoperazone. A final pattern emerges when the cefotaxime comparison is considered. The innate antigram-negative activity translates well between the two compounds; however the monobactam exhibits poor antigram-positive activity and demonstrably is less stable to β-lactamase.

These studies underscore the unique nature of β-lactam nuclei. Although they all share a common target, i.e. inhibition of bacterial cell wall synthesis, structural modification may subtly or drastically affect the ability of a molecule to interact with receptor sites, gain access to the receptor sites, or withstand the onslaught of β-lactamase. While the penicillin and cephalosporin side chains provided a general overview of the range of possible activities, they were only a start in the systematic investigations on this novel nucleus.

After examination of a wide range of penicillin and cephalosporin surrogates, attention focused on a particular class of side chains, the aminothiazole oximes, side chains common to many of the third-generation cephalosporins.

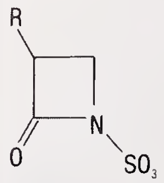
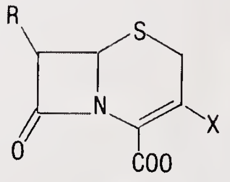
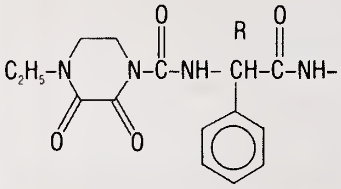
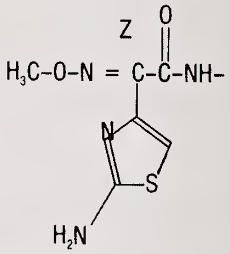
Examination of Table 3 reveals the remarkable gain in activity accompanying introduction of a methoxyimino function in concert with a 2-aminothiazol-4-ylacetyl side chain. SQ 81,377, a monobactam analog of the third-generation cephalosporin cefotaxime, shows a similar spectrum of activity. Unlike cefotaxime however, SQ 81,377 is unstable to many β-lactamases elaborated by gram-negative bacteria. Further structural modification of the nucleus revealed that 4-methylation could have a profound effect on β-lactamase stability.

Table 4 illustrates the effect of 4-methylation on β-lactamase stability. This increased stability is mirrored in the increase in activity noted for these methylated compounds against β-lactamase producing gram-negative bacteria (Table 5). The comparison of MIC values at low and high inoculum levels especially with reference to the three paired sets of organisms, which are β-lactamase and non-β-lactamase producing strains, makes this point evident. Detailed consideration of these and similar results led to the choice of aztreonam (SQ 26,776) for clinical development.

The structure-activity relationships in the aztreonam molecule are shown in Figure 6. From this diagram we can see the influence of the various substituents attached to the monocyclic β-lactam ring. The

TABLE 3

Antibacterial Relationships of Cephalosporins to Their Homologous Monobactams
*Minimum Inhibitory Concentrations (μg/ml): Agar Dilution Assays**

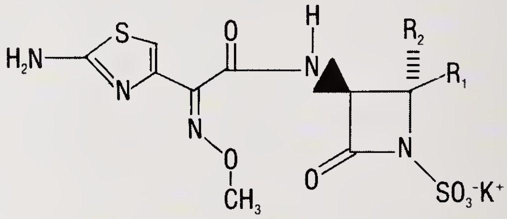
 *Monobactam*	 *Cephalosporin*		**R**								
			Nucleus	**Cephalosporin**		**Monobactam**		**Cephalosporin**		**Monobactam**	
			Test Compounds	**Cefoperazone**		**SQ 81,491**		**Cefotaxime**		**SQ 81,364**	
			CFU	**10^4**		**10^4**		**10^4**		**10^4**	
Organism		**SC Number**									
Staphylococcus aureus	PENase-	2,399		0.8		3.1		0.8		6.3	
Staphylococcus aureus	PENase+	2,400		1.6		3.1		0.8		6.3	
Escherichia coli		8,294		0.8		0.4		0.1		0.8	
Klebsiella aerogenes		10,440		0.8		0.4		0.1		0.8	
Proteus rettgeri		8,479		0.1		>0.05		>0.05		>0.05	
Enterobacter cloacae		8,236		0.8		0.8		0.4		0.8	
Pseudomonas aeruginosa		8,329		3.1		3.1		12.5		12.5	
			CFU	**10^4**	**10^6**	**10^4**	**10^6**	**10^4**	**10^6**	**10^4**	**10^6**
Escherichia coli	TEM+	10,404		3.1	>100	12.5	>100	0.05	0.1	6.3	>100
Escherichia coli	TEM-	10,439		0.2	0.4	0.4	0.4	>0.05	0.1	0.8	1.6
Enterobacter cloacae	P99+	10,435		50	>100	>100	>100	50	>100	>100	>100
Enterobacter cloacae	P99-	10,441		0.2	0.4	0.2	0.8	0.2	0.2	1.6	3.1
Klebsiella aerogenes	K1+	10,436		>100	>100	>100	>100	1.6	100	>100	>100
Klebsiella aerogenes	K1-	10,440		0.1	0.2	0.48	6.3	>0.05	>0.05	0.8	0.8

*Minimum inhibitory concentrations were determined by a twofold agar dilution method on DST agar.

sulfonic acid grouping attached to the nitrogen of the β-lactam ring is responsible for "activating" the β-lactam carbonyl. The α-methyl group at position 4 enhances the stability of the ring to β-lactamase attack, in addition to providing increased antibacterial activity. The aminothiazole oxime moiety on the acyl side chain is responsible for the potent activity exhibited by aztreonam against aerobic gram-negative bacteria. Activity against *Pseudomonas* is enhanced by the addition of two methyl groups and a carboxylic acid function on the oxime side chain. These various attributes of potent activity against a wide range of gram-negative bacteria including *Pseudomonas aeruginosa*, allied with limited potency against gram-positive bacteria and anaerobes, introduce a unique spectrum of activity among the β-lactam antibiotics.

TABLE 4

β-Lactamase Stability of Monobactams



*β-Lactamase Stability**

R_1	R_2	TEM	P99	K1
H	H	12	0.08	106
CH_3	H	0.3	<0.02	6.4
H	CH_3	1	<0.07	20

*Cephaloridine = 100

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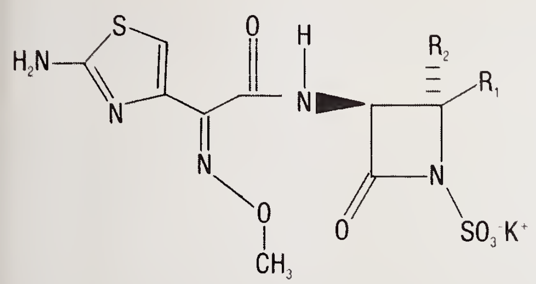
TABLE 5

4-Methylated Monobactams

 H2N S N O H N R2 R1 N O CH3 O N SO3⁻K⁺		R₁	H		CH₃		H	
		R₂	H		H		CH₃	
MIC (μg/ml) Agar Dil. Innocula (CFU)			**10^4**	**10^6**	**10^4**	**10^6**	**10^4**	**10^6**
Organism	**SC No.**	**SQ No.**	81,377		28,863		26,668	
E. coli TEM+	10,404		6.3	>100	0.1	0.1	0.4	1.6
E. coli TEM-	10,439		0.4	1.6	<0.05	0.1	0.1	0.2
Ent. cloacae P99+	10,435		>100	>100	3.1	12.5	25	25
Ent. cloacae P99-	10,441		1.6	1.6	0.1	0.2	0.2	0.4
K. aerogenes K1+	10,436		>100	>100	12.5	>100	>100	>100
K. aerogenes K1-	10,440		0.4	1.6	<0.05	0.1	0.2	0.2
K. pneumoniae	11,066		12.5	100	0.1	0.2	1.6	12.5
C. freundii	10,204		0.4	>100	0.1	0.2	0.8	1.6
Prot. rettgeri	8,217		1.6	25	0.1	0.2	0.1	0.4
Prot. vulgaris	10,951B		100	>100	<0.05	0.1	0.4	50
Ser. marcescens	9,782		25	50	0.2	1.6	0.8	3.1
P. aeruginosa	8,329		12.5	25	>100	>100	>100	>100
P. aeruginosa	9,545		0.8	12.5	0.8	3.1	0.8	0.8

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AZTREONAM: DISPOSITION AND PHARMACOKINETICS IN RATS, DOGS, AND MONKEYS

BRUCE H. MIGDALOF, PH.D.*

The distribution, metabolism, and excretion of aztreonam, a parenterally administered novel monocyclic β-lactam antibiotic, was studied in rats, dogs, and monkeys. The biological fate (biotransformation and excretion) of aztreonam was similar to that of other β-lactam antibiotics.

Since aztreonam (Figure) is the first of the newest class of β-lactam antibiotics, the monocyclic β-lactams or monobactams, we were interested not only in elucidating the fate of aztreonam in vivo, but also in a comparison of the biotransformation of aztreonam with the published biotransformation information on previously studied β-lactam antibiotics, i.e. penicillins and cephalosporins. All our studies were carried out using aztreonam labeled with ^{14}C in the thiazole ring of the side chain. The deactivation of β-lactam antibiotics by micro-organisms is accomplished primarily by the action of β-lactamases. Although mammalian cells are devoid of β-lactamase activity, chemical (nonenzymatic) hydrolysis of the β-lactam moiety can occur in biological systems. Indeed, compounds arising from hydrolysis of the β-lactam ring of β-lactam antibiotics can be present as minor impurities in the antibiotic. The term "biotransformation product" will be used to describe the compounds formed from the parent molecule in vivo whether by enzymatic (metabolic) processes or by nonenzymatic (chemical) processes. Results obtained after parenteral administration of ^{14}C-aztreonam to rats, dogs, and monkeys will be presented.

DISPOSITION IN RATS

When ^{14}C-aztreonam was administered as single 50 mg/kg intramuscular (IM) or intravenous (IV) doses to male CD outbred albino rats,[1] total recovery of the radioactive doses in 96 hr averaged (mean±SEM; n=5) 71 ± 5 percent (IM) and 68 ± 2 percent (IV) in urine and 26 ± 2 percent (IM) and 33 ± 3 percent (IV) in feces.

Biotransformation profiles of radioactivity in urine were generated using thin-layer radiochromatography (TLRC). Profiles after IM and IV doses were essentially identical; unchanged ^{14}C-aztreonam accounted for 83 ± 1 percent (IM) and 85 ± 1 percent (IV) of the radioactivity in the urine collected in the first 24 hr after dosing (57 percent of the IM or IV dose). The most prominent biotransformation product in urine accounted for 14 ± 1 percent (IM) and 13 ± 1 percent (IV) of the radioactivity in urine (9 to 10 percent of the dose) and was identified as SQ 26,992, the compound formed by hydrolysis of the β-lactam ring (Figure). The remainder of the radioactivity in urine (only about 2 percent of the dose) was attributed to unidentified biotransformation products.

DISTRIBUTION IN RAT TISSUES

The distribution of total radioactivity (^{14}C-aztreonam and all biotransformation products collectively) and unchanged ^{14}C-aztreonam in tissues was determined in groups of male and female CD outbred albino rats (five of each sex at each time point) 0.25, 2, 6, and 24 hr after single 50 mg/kg doses of ^{14}C-aztreonam adminstered, with an equimolar amount of L-arginine, intramuscularly into the quadriceps femoris.[2]

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In comparing concentrations of radioactivity in tissues common to both sexes, no major differences were observed at any of the time points studied. Concentrations of radioactivity were, in general, highest at the first time point (0.25 hr) and then declined to low but detectable (≥ 0.01 μg equivalents of aztreonam equivalents per ml) concentrations at 24 hr. In general, at 24 hr, concentrations in serum exceeded those in tissues; exceptions were tissues related to excretory functions [kidneys, large intestine and contents, liver, small intestines (females only), stomach, and urinary bladder], meninges, and lymph nodes (females only). Whole body autoradiographic studies confirmed the results of the analyses of selected tissues.

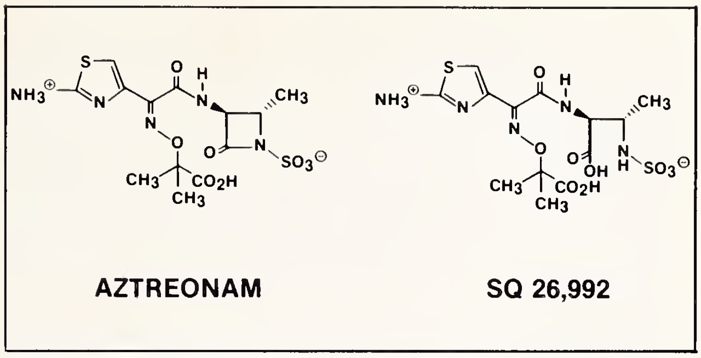
In selected tissues (serum, kidney, liver, lung, small intestine/contents, and large intestine/contents), concentrations of unchanged aztreonam were determined by TLRC of methanolic extracts (detection limit: 0.01 μg/ml). At 2 hr after the IM dose, concentrations of aztreonam in serum averaged 8.7 ± 2.6 μg/ml (males) and 15 ± 4.5 μg/ml (female). At 2 hr, concentrations of aztreonam were higher in the small intestine and its contents than in the large intestine and its contents, whereas at 6 hr and 12 hr concentrations in large intestine and its contents were higher. In males and females, concentrations of aztreonam in kidneys and lungs decreased at a slower rate than in serum; concentrations in liver decreased at approximately the same rate as in serum. In target tissues where concentrations of aztreonam declined more slowly than in serum, aztreonam may exhibit a longer duration of action than would be suggested by serum concentration data.

PLACENTAL TRANSFER IN RATS

Placental transfer was investigated[3] on day 16 of gestation by analyzing samples of maternal blood, fetuses, placentas, and amniotic fluid obtained one, two, four, and eight hr after single doses of ^{14}C-aztreonam were administered subcutaneously (SC) to female (CD) outbred rats at 150 mg/kg (a dose given daily in teratological and reproductive studies of aztreonam). Maximum concentrations of total radioactivity were attained within one hr in all tissues. In general, concentrations of total radioactivity in maternal blood exceeded the concentrations in the other three tissues. Only the placenta at eight hr had a mean concentration that exceeded the mean concentration in serum at that time. The mean maternal serum: tissue ratios of concentrations for both total radioactivity and unchanged aztreonam changed with time in a manner that reflected a slower decline of concentrations in the three tissues than in maternal serum.

EXCRETION IN MILK OF LACTATING RATS

To compare concentrations of ^{14}C-aztreonam and its metabolites in milk and maternal serum, dams were given single 150 mg/kg SC doses of ^{14}C-aztreonam on day 14 of lactation.[3] Milk samples were obtained from groups of dams at either one and four hr or at two and eight hr after dosing with ^{14}C-aztreonam. The highest concentrations of total radioactivity and unchanged aztreonam observed were at one hr. Concentrations of total radioactivity and of unchanged aztreonam declined more slowly in milk than in serum.



Figure—Structure of aztreonam and its biotransformation product, SQ 26,992.

DISPOSITION IN DOGS

Single 25 mg/kg doses of ^{14}C-aztreonam were administered to four male purebred beagle dogs SC and IV.[1] Recovery of the radioactive doses in 96 hr averaged (mean ± SEM; n=4) 90 ± 2 percent (SC) and 86 ± 2 percent (IV) in urine and averaged 9 ± 1 percent (SC) and 7 ± 1 percent (IV) in feces. Aztreonam was apparently released relatively slowly from the SC injection site; excretion in urine in 2 hr after dosing averaged 67 percent after the IV dose, but only 24 percent after the SC dose.

After an SC or IV dose, 85 to 86 percent of ^{14}C-radioactivity in 0 to 24 hr urine samples was attributable to unchanged ^{14}C-aztreonam, 10 to 11 percent SQ 26,992, and 4 percent was present as unidentified compounds. Unchanged aztreonam excreted in the urine in 24 hr accounted for 75 ± 4 percent of the SC dose and 73 ± 2 percent of the IV dose.

After SC injection, maximum concentrations of total radioactivity in serum were observed between 0.5 and 1.0 hr (T_{max} = 0.9 ± 0.1 hr; n=4) and ranged from 33 to 36 μg/ml (C_{max} = 35 ± 1 μg/ml). Commencing at 1 hr and extending throughout the remainder of the 96-hr study period, concentrations of radioactivity in serum were greater after the IM than after the IV dose. The mean AUC values (0 to 96 hr) for total radioactivity in microgram-hours per ml of aztreonam equivalents (mean ± SEM; n=4) were 149 ± 5 (IV) and 168 ± 21 (SC); the difference was not statistically significant. The higher concentrations of radioactivity in serum after 1 hr for the SC dose when compared with concentrations after the IV dose suggest that absorption from the SC site occurred over time (i.e. was not instantaneous); AUC values indicated absorption of the SC dose was complete.

Maximum concentrations of unchanged aztreonam in serum ranged from 13 to 19 μg/ml after the SC dose (C_{max} = 17 ± 1 μg/ml) and were attained between 0.5 and 1 hr (T_{max} = 0.9 ± 0.1 hr). Between 1 and 6 hr, concentrations of unchanged aztreonam were higher after SC than after IV dosing, due to release from the SC dose site over time. The AUC values (0 to 8 hr) averaged 44 ± 3 μg x hr/ml (IV) and 49 ± 4 μg x hr/ml (SC); the values were not statistically significantly different. The apparent serum elimination half-life ($t_{1/2\beta}$) for unchanged aztreonam averaged 0.7 ± 0.01 hr (IV dose) and 1.3 hr (SC dose; n=2). The apparently longer $t_{1/2\beta}$ after the SC dose was a reflection of the rate of absorption of the SC dose from the site of dosing. Total radioactivity in serum not accounted for as aztreonam was present (in decreasing order) as components unextractable with methanol, as SQ 26,992 and as other extractable but unidentified biotransformation products.

Serum protein binding for total radioactivity was determined at 0.25 and 2.0 hr; no time-dependency was observed. The serum protein binding (overall for 0.25 and 2 hr) averaged 28 ± 3 percent (IV) and 34 ± 3 percent (SC); the difference was not statistically significant.

DISPOSITION IN MONKEYS

Four female cynomolgus monkeys each received single 25 mg/kg doses of ^{14}C-aztreonam IV and IM.[1] Recovery of the radioactive doses in 96 hr after dosing averaged (mean ± SEM; n=4) 43 ± 2 percent (IM) and 44± 6 percent (IV) in urine, and 43 ± 5 percent (IM) and 46 ± 3 percent (IV) in feces. Recovery was incomplete due, in part, to excretion continuing past the end of the collection period; after an IM or IV dose, about 2 percent of the radioactive dose was excreted in the last collection interval (72 to 96 hr). About half of the radioactivity recovered in urine was excreted in the first 4 hr; 23 ± 7 percent of the dose after IM and 25 ± 5 percent after IV dosing.

Unchanged aztreonam accounted for 78 ± 4 percent (IM) and 77 ± 1 percent (IV) of the radioactivity in 0- to 24-hr urine. After IM and IV administration, the relative percentage of radioactivity in urine attributable to unchanged aztreonam decreased from about 88 percent in 0- to 4-hr urine to about 25 to 30 percent in 8- to 24-hr urine. SQ 26,992, accounted for 14 to 15 percent (IM) and 12 to 13 percent (IV) of the radioactivity in 0- to 24-hr urine and accounted for a higher percentage of the radioactivity in urine in 8 to 24 hr than in 0 to 4 hr. Three other minor biotransformation products present in urine were isolated and found to be devoid of detectable antimicrobial activity; these biotransformation products were not further characterized or identified, but their HPLC retention times indicated that they were more polar than SQ 26,992. In monkey feces, SQ 26,992 was the major radioactive component after an IM and IV dose of ^{14}C-aztreonam. In the 0- to 72-hr fecal samples, SQ 26,992 accounted for 47 ± 3 percent (IM) and 46 ± 3 percent (IV) of the radioactivity, and unchanged aztreonam for 11 ± 1 percent (IM) and 17 ± 4 percent (IV). Other biotransformation products in feces were not characterized further.

Studies used aztreonam labeled with ^{14}C in the thiazole ring of the side chain.

Concentrations of radioactivity in serum from 0.5 to 24 hr after single IM and IV doses were comparable. Selected samples (0.5, 1, and 4 hr) were analyzed for unchanged aztreonam; results were similar after IM and IV doses. At 1 hr, concentrations of total radioactivity in serum averaged 37 ± 3 μg-equivalents/ml (IM) and 39 ± 2 μg-equivalents/ml (IV); unchanged aztreonam concentrations at 1 hr averaged 26 ± 2 μg/ml (IM) and 30 ± 2 μg/ml (IV).

Binding of total radioactivity to serum proteins at 0.25 and 2 hr averaged 49 ± 1 percent and 56 ± 3 percent (IM) and 49 ± 1 percent and 59 ± 3 percent (IV); differences between values obtained from IM and IV doses were not statistically significant.

DISPOSITION OF AZTREONAM RELATIVE TO OTHER β-LACTAM ANTIBIOTICS

The question as to whether the unique monocyclic β-lactam structure of aztreonam would be reflected in atypical disposition was addressed by comparing our disposition data for aztreonam with data (for animals and humans) from published literature on classic β-lactam antibiotics including penicillins such as penicillin V, phenethicillin, oxacillin, benzylpenicillin, ampicillin, and amoxycillin and cephalosporins such as cefotaxime and çephatrizine.[4-7] In the penicillin studies reported, between 80 percent and 100 percent of a single dose of the antibiotic was excreted in urine. Biotransformation of the penicillins was minimal; most were excreted unchanged in urine. The predominant biotransformation product of the penicillins accounted for 2 to 15 percent of the dose and was the penicilloic acid which resulted from hydrolysis of the β-lactam ring. The penicilloic acids were more slowly excreted than the penicillin parent compound.[4] For cephalosporins, metabolism also was minimal; excretion in urine was extensive and the biotransformation product resulting from hydrolysis of the β-lactam ring was reported. The disposition of aztreonam, the first monobactam, in rats, dogs, and monkeys has been discussed. The disposition of ^{14}C-aztreonam was studied in normal subjects and the results were quantitatively similar to that found in rats, dogs, and monkeys. In 144 hr after a single dose of ^{14}C-aztreonam (500 mg), excretion averaged 76 percent (IV) and 78 percent (IM) in urine and 12 percent (IV) and 15 percent (IM) in feces.[8] The biotransformation profile in 0 to 48 hr urine indicated that 88 percent of the radioactivity in urine (66 to 67 percent of the dose; IV or IM) was excreted as unchanged aztreonam and 10 percent (7 percent of the dose; IV or IM) as SQ 26,992. Unidentified biotransformation products accounted for the remaining 3 percent of the radioactivity in urine (about 2 percent of the dose).

A comparison of our data on aztreonam with published reports on other β-lactam antibiotics indicates that the excretion and biotransformation of aztreonam followed the same general pattern as seen for other β-lactam antibiotics. Excretion of the unchanged β-lactam antibiotic in urine was the predominant elimination pathway, with minor amounts of the dose excreted more slowly in the form of a biotransformation product arising from hydrolysis of the β-lactam ring. Where other minor metabolites were reported, they generally were devoid of antimicrobial activity.

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AZTREONAM: PRECLINICAL STUDIES

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Aztreonam specifically is targeted against the problem of gram-negative bacteria responsible for the majority of nosocomial infections. Aztreonam offers the opportunity for a more rational approach in the therapy of these infections.

The development of aztreonam (Figure 1) came about through a recognition of the changing patterns of bacterial diseases and the increasing sophistication in the therapeutic approaches used for their remedy. In recent years, nosocomial infections caused by multiply resistant aerobic gram-negative bacteria have emerged as a major clinical problem. The entry of soil organisms such as *Pseudomonas*, *Enterobacter*, and *Serratia* into the hospital along with resistance development in more traditional gram-negative pathogens, such as *Escherichia coli* and *Klebsiella pneumoniae*, have challenged the usefulness of the early penicillins and cephalosporins. In addition, a shift in patient population represented by an increase in older individuals with multiple underlying diseases or compromised immune systems due to cancer chemotherapy or transplantation has forced a reconsideration of prescribing habits. Finally, the growth of the clinical laboratory where the infectious etiologic agent can be identified more readily and rapidly and its antibiotic susceptibilities reliably determined has allowed for a more rational approach to antibiotic therapy. An awareness of these changes was influential in the decision to develop aztreonam for clinical use.

Aztreonam specifically is directed against aerobic gram-negative bacteria and has limited activity against gram-positive bacteria and anaerobes. Despite a novel structure and unique activity spectrum, aztreonam acts like other β-lactams through interference with bacterial cell wall biosynthesis. The selectivity of aztreonam for the essential penicillin-binding proteins (PBPs) of bacteria accounts for its differential activity spectrum. The relative inactivity for gram-positive and anaerobic bacteria derives from a poor interaction of aztreonam with the PBPs of these organisms. A very high affinity for the septum peptidoglycan transpeptidase (PBP3) of gram-negative bacteria results in potent activity against this group of organisms (Table 1). In binding to PBP3 of gram-negative bacteria, aztreonam causes filamentation of the affected organism, a morphological response identical to that observed with most of the cephalosporins.[1] Rupture of the cell wall follows filamentation and the ensuing lysis results in death of the bacterium.[2]

The other major enzyme system with which β-lactam antibiotics often interact are the β-lactamases. This diverse array of hydrolytic enzymes accounts for most of the resistance to β-lactams commonly seen in pathogenic bacteria. The β-lactamases may be plasmid or chromosomally mediated and their widespread distribution has been a constraining factor on the utility of susceptible β-lactams. The plasmid-mediated β-lac-

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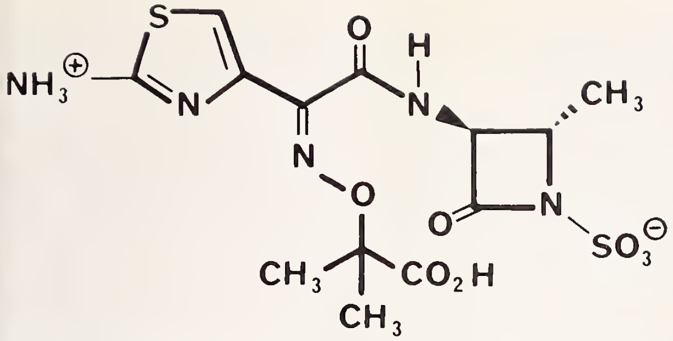

Figure 1—Structure of aztreonam.

tamases are particularly insidious due to the promiscuous behavior of the plasmids. For example, plasmids carrying the genes responsible for TEM β-lactamase have the ability to move freely among the *Enterobacteriaceae*, pseudomonads, *Neisseria gonorrhoeae*, and *Hemophilus influenzae*. The β-lactamases that are chromosomally mediated are restricted in their mobility under normal circumstances, but frequently are found in species of *Enterobacter*, *Serratia*, *Proteus*, and *Pseudomonas*. It is not unusual in certain organisms to find both plasmid and chromosomally mediated enzymes. Aztreonam shows unusual stability to the common plasmid and chromosomally mediated β-lactamases of gram-negative bacteria, such as the TEM and OXA series and the Class 1 enzymes (Tables 2 and 3). Only the PSE-2 β-lactamase among the plasmid enzymes shows some slight hydrolysis of aztreonam. Among the chromosomal-directed enzymes, only the relatively uncommon K1 β-lactamase produced by certain strains of *Klebsiella oxytoca* hydrolyzes aztreonam to a moderate extent.

In addition to their inherent hydrolytic ability, many of the chromosomally mediated β-lactamases are inducible enzymes. In the presence of β-lactam antibiotics their production markedly may be increased leading to elevated concentrations strategically localized in the periplasmic space.[3] Aztreonam is unique in that it does not induce increased production of the chromosomally mediated enzymes and consequently must deal only with basal levels.[4] Good inducers such as cefoxitin may be at times compromised when the stability to the elicited β-lactamase is marginal.

Aztreonam with its discriminating activity spectrum is unique among the β-lactam antibiotics. Specifically directed against aerobic gram-negative bacteria, aztreonam shows high activity against members of the *Enterobacteriaceae*, *Hemophilus*, *Neisseria*, and *P. aeruginosa* (Table 4). The absence of clinically meaningful activity against gram-positive and anaerobic bacteria completes the selective antibacterial profile. Against the enteric bacteria, aztreonam is superior to cefotaxime having MIC_{90} values of less than 1.0 mcg/ml except for the *Enterobacter* group. Most strains of *Enterobacter* also were highly susceptible to aztreonam with 80 percent being inhibited by 1.0 mcg/ml or less. The presence of a few permeability mutants skews the susceptibility curve against both aztreonam and cefotaxime. With strains of

TABLE 1

*Binding of Aztreonam to PBPs of Different Bacteria**

		Concentration (μg/ml) for Complete Inhibition of Benzylpenicillin Binding									
Organism		**PBP1**	**PBP1a**	**PBP1b**	**PBP1c**	**PBP2**	**PBP3**	**PBP4**	**PBP5**	**PBP5/6**	**MIC (μg/ml)**
E. coli	SC 8294**		10	≥100		>100	0.1	100		100	0.1
P. mirabilis	SC 3855		2.0	100		>100	0.1	100		>100	<0.1
P. vulgaris	SC 9416		10	≥100		ND	0.1	ND		>100	<0.05
K. pneumoniae	SC 9527		ND†	>100		>100	0.1	>100		>100	0.1
P. aeruginosa	SC 9545		ND	100	30	>100	0.1	>100		>100	0.2
S. aureus	SC 2399	>100				≥100	>100	>100			>100
B. fragilis	SC 11085	>100				100	ND	>100	>100		>100

*From Georgopapadakou et al. (1982).[1]
**Squibb Culture Collection Number
†Not Detected

TABLE 2

*Stability of Aztreonam to Plasmid-Mediated β-Lactamases from Gram-Negative Bacteria**

β-Lactamese	Relative Efficiency of Hydrolysis			
	Aztreonam	**Cefotaxime**	**Cefoperazone**	**Cephaloridine**
TEM-1	0.04	0.1	70	100
TEM-2	0.03	0.07	60	100
OXA-2	1.0	6.0	250	100
SHV-1	0.1	0.14	150	100
PSE-1	0.02	0.03	260	100
PSE-2	5.5	40	1900	100
PSE-3	0.8	0.3	6	100
PSE-4	0.01	0.03	280	100

*From Sykes et al. (1982).[6]

TABLE 3

*Stability of Aztreonam to Chromosomally Mediated β-Lactamases from Gram-Negative Bacteria**

β-Lactamase	Relative Efficiency of Hydrolysis			
	Aztreonam	**Cefotaxime**	**Cefoperazone**	**Cephaloridine**
Klebsiella K1	10	5.0	30	100
Proteus P1	<1.0	0.6	>1	100
Serratia S1	<0.2	5.0	30	100
Enterobacter P99	<0.01	5.0	7.0	100
Enterobacter E1	<0.01	0.02	5.0	100
Providencia PD1	0.03	3.0	5.0	100
Bacteriodes B1	1.1	30	40	100

*From Sykes et al. (1982).[6]

Hemophilus influenzae and *Neisseria gonorrhoeae*, both aztreonam and cefotaxime were inhibitory at very low levels. In the case of *P. aeruginosa*, aztreonam is three- to fourfold more active than cefotaxime and consequently has MIC values in the range of clinical utility. The studies with *P. aeruginosa* were expanded by testing the susceptibility of 45 clinical strains against recently approved β-lactam antibiotics (Table 5). Aztreonam shows a clear superiority to cefotaxime, ceftizoxime, and moxalactam. Cefoperazone and piperacillin, on the other hand, are similarly inhibitory to aztreonam against these routine isolates. When specific β-lactamase-producing strains of *P. aeruginosa* are considered, however, the inherent enzyme instability of cefoperazone and piperacillin is manifest in elevated MIC values while the inhibitory activity of aztreonam is unaffected (Table 6). This provides confirmation of the results obtained in studies with the cell-free β-lactamase preparations.

Clinical strains of bacteria specifically resistant to other antibiotics in common use also have been investigated. For example, 120 enteric bacteria resistant to cefamandole were tested for susceptibility to aztreonam. Aztreonam inhibited 50 percent at less than 0.2 mcg/ml and 90 percent at less than 6.3 mcg/ml (Figure 2). Similarly when 46 gentamicin-resistant isolates were examined, aztreonam inhibited 50 percent at 0.2 mcg/ml and 90 percent at less than 3.1 mcg/ml (Figure 3). These two examples not only underscore the widespread problems of resistance among the *Enterobacteriaceae* but demonstrate the potential of aztreonam to deal with these problems.

Another area of recent interest has been the increasing use of combination antibiotic therapy in the seriously ill patient. Such an approach seeks not only to take advantage of potential synergistic interactions between the administered antibiotics but also to provide sufficiently broad antibacterial coverage against the etiologic agent when its identity and susceptibility are unknown. Studies have been carried out with aztreonam addressing both situations.

In the empiric situation, initial therapy may involve aztreonam in combination with an agent active against gram-positive bacteria. Once the pathogen has been identified as a gram-positive or gram-negative bacterium the inappropriate antibiotic would be discontinued. To insure microbiological compatibility, in vitro studies have been conducted on aztreonam in combination with a number of antibiotics offering primarily a gram-positive spectrum. When tested with

TABLE 4

*Antibacterial Spectrum of Aztreonam**

Organism (no. of strains)	MIC_{90} (μg/ml)** **Aztreonam**	 **Cefotaxime**
Staphylococcus aureus (12)	>100	3.1
Streptococcus pyogenes (11)	12.5	<0.1
Streptococcus pneumoniae (11)	>100	<0.1
Streptococcus faecalis (12)	>100	>100
Bacteroides fragilis (8)	>100	>100
Escherichia coli (79)	0.2	0.2
Klebsiella pneumoniae (68)	0.3	0.3
Enterobacter cloacae (29)	12.5	25.0
Enterobacter aerogenes (13)	33.3	42.9
Serratia marcescens (113)	1.6	8.4
Proteus mirabilis (25)	<0.1	<0.1
Proteus vulgaris (11)	<0.1	5.5
Proteus rettgeri (6)	<0.1	0.2
Proteus morganii (19)	0.6	1.6
Providencia stuartii (15)	<0.1	3.0
Salmonella sp (25)	0.3	0.3
Citrobacter freundii (25)	0.7	0.4
Acinetobacter calcoaceticus (25)	58.3	24.4
Pseudomonas aeruginosa (61)	12.0	47.8
Hemophilus influenzae Amp^s (18)	0.2	<0.1
Hemophilus influenzae Amp^r (18)	0.2	<0.1
Neisseria gonorrhoeae (20)	0.2	<0.1

*From Sykes et al. (1982).[6]

**MIC_{90} (μg/ml): Concentration of antibiotic necessary to inhibit 90 percent of strains tested.

nafcillin, cloxacillin, erythromycin, or vancomycin, aztreonam had no adverse effects on the activity of these antibiotics against gram-positive bacteria.[5] Conversely, when gram-negative bacteria were tested, the added antibiotics had no adverse effects on the activity of aztreonam. Additivity or indifference was the usual response of the organism to the combination.

TABLE 5

Activity of Aztreonam and Other β-Lactams v 45 strains of P. aeruginosa

Test Compounds	**MIC_{50} (μg/ml)***	**MIC_{90} (μg/ml)***
Aztreonam	5.6	12.4
Moxalactam	21.7	44.5
Cefotaxime	14.0	45.9
Ceftizoxime	33.6	91.8
Cefoperazone	5.5	11.9
Piperacillin	5.3	14.6

*Minimum inhibitory concentrations for 50 percent and 90 percent of the strains tested. MIC's determined by serial twofold agar dilution assays in Diagnostic Sensitivity Test Agar (Oxoid) at 5 x 10^5 colony-forming units.

In marked contrast, a very positive interaction may occur with aztreonam-aminoglycoside combinations against certain groups of organisms. Against isolates of *P. aeruginosa*, high incidence of synergy is found when aztreonam is combined with gentamicin, tobramycin, or amikacin (Table 7). Synergy also is seen against strains of *Acinetobacter* but at a frequency lower than that observed with the pseudomonads. The synergistic interactions of the aztreonam-aminoglycoside combinations are equally impressive when aminoglycoside-resistant enteric bacteria are tested (Table 8). Depending on the particular combination and group of organisms tested, the incidence of synergy has ranged upwards to 80 percent. The import of this synergy or increased potency readily is apparent especially when considering the granulocytopenic patient with a gram-negative bacterial infection.

The properties of aztreonam established in vitro have translated well in a number of animal model infections. In murine systemic infections involving gram-negative bacteria, aztreonam has shown efficacy equal or superior to third-generation cephalosporins.[6] In mouse, rat, and rabbit infections where pathogens have been localized in distinct anatomic sites,

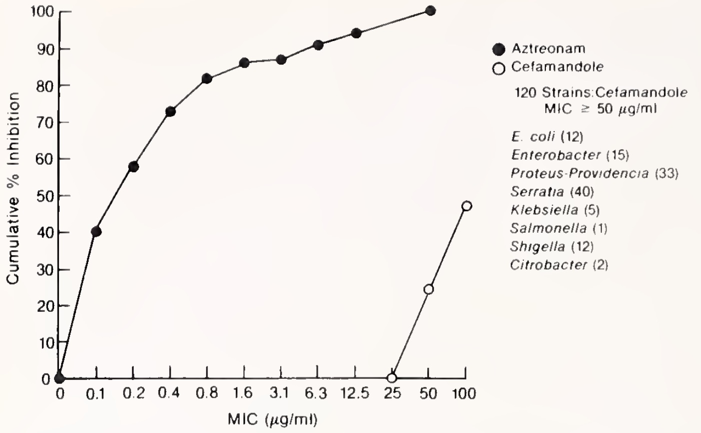

Figure 2—Activity of aztreonam *v* cefamandole-resistant bacteria.

aztreonam also has demonstrated good efficacy. Wound infections in mice,[7] meningitis in rats,[7] and endocarditis in rabbits[8] involving a variety of gram-negative bacteria all have responded to aztreonam therapy. Even in the neutropenic situation involving compromised rats infected with *K. pneumoniae*, aztreonam has been proved effective.[9]

The selective antibacterial spectrum of aztreonam confers an added positive attribute in minimizing the deleterious effects on the indigenous host microflora.

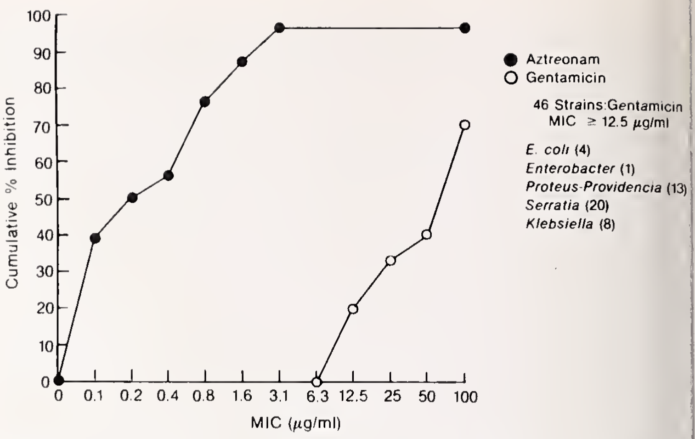

Figure 3—Activity of aztreonam *v* gentamicin-resistant bacteria.

Studies in hamsters have established that aztreonam with its lack of activity against anaerobic and gram-positive bacteria does not elicit the massive shifts in the bacterial makeup of the gastrointestinal microflora seen with broad spectrum antibiotics. In preserving the more natural state, the resident micro-organisms can fulfill their normal functions of providing essential body nutrients and presenting a barrier to colonization and invasion by potential pathogens. This has been exemplified in the hamster model for hemor-

TABLE 6

Activity of Aztreonam, Cefoperazone, and Piperacillin v β-Lactamase Producing Strains of P. aeruginosa.

Strain Number	β-Lactamese	MIC (μg/ml)* Aztreonam	Cefoperazone	Piperacillin
SC 12126	OXA-2	25	>100	>100
SC 12136	OXA-3	12.5	>100	>100
SC 12128	PSE-1	12.5	>100	>100
SC 12138	PSE-2	12.5	>100	>100
SC 12137	PSE-3	12.5	25	>100
SC 12130	PSE-4	12.5	>100	>100
SC 12135	TEM-1	6.3	>100	>100
SC 12133	TEM-2	1.6	>100	>100
SC 12131	SHV-1	12.5	>100	>100

*Minimum inhibitory concentration determined by serial twofold agar dilution assays in Diagnostic Sensitivity Test Agar (Oxoid) at 10^6 colony-forming units.

TABLE 7

Interaction of Aztreonam with Aminoglycosides Against Gram-Negative Bacteria

Group	Number of Strains Synergistically Inhibited* (number/total tested) Aztreonam/ Gentamicin	Aztreonam/ Tobramycin	Aztreonam/ Amikacin
P. aeurginosa	19/27	24/27	22/27
Acinetobacter	6/25	7/25	8/25

*Synergy defined as FIC ≤0.5: a fourfold reduction in MIC for each antibiotic when tested together as compared to the MIC's obtained when the antibiotics were tested separately.

TABLE 8

Interaction of Aztreonam with Aminoglycosides Against Aminoglycoside-Resistant Gram-Negative Bacteria

Group	Number of Strains Synergistically Inhibited* (number/total tested)		
	Aztreonam/ Gentamicin	**Aztreonam/ Tobramycin**	**Aztreonam/ Amikacin**
Gentamicin-resistant**	12/23	16/23	16/23
Tobramycin-resistant+	11/22	16/22	15/22
Amikacin-resistant°	4/10	0/10	6/10

*Synergy defined as FIC ≤0.5: a fourfold reduction in MIC for each antibiotic when tested together as compared to the MIC's obtained when the antibiotics were tested separately.

***E. coli* (2), *Serratia* (8), *Klebsiella* (4), *Enterobacter* (3), *Providencia* (4), *Morganella* (1), *Pseudomonas* (1).

+*E. coli* (2), *Serratia* (8), *Klebsiella* (4), *Enterobacter* (3), *Providencia* (4), *Morganella* (1), *Pseudomonas* (1).

°*E. coli* (2), *Serratia* (5), *Providencia* (3).

rhagic caecitis which is considered analogous to antibiotic-associated colitis in man.[10,11] Administration of a broad-spectrum antibiotic such as cefoperazone to hamsters results in a substantial decrease in the anaerobic flora of the gastrointestinal tract followed by colonization and proliferation of *Clostridium difficile*, elaboration of the *C. difficile* cytotoxin, hemorrhagic caecitis, and death. Administration of aztreonam does not cause these changes and the hamsters remain healthy for the duration of the experiment.[12]

In the preclinical development of aztreonam, an antibiotic profile has emerged that is contemporary with the increasingly sophisticated approaches being taken in the therapy of bacterial disease. Although narrow in spectrum, aztreonam specifically is targeted against the problem gram-negative bacteria responsible for the majority of nosocomial infections. Aztreonam with its directed spectrum offers the opportunity for a more rational approach in the therapy of these infections. As an alternative to broad-spectrum antibiotics, aztreonam should minimize some of the side effects seen with these agents and lead to a better maintenance of the host defensive microflora and a moderation of the rapid emergence of resistant bacteria.

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AZTREONAM: EARLY CLINICAL STUDIES IN NEW JERSEY

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Studies of efficacy and safety of aztreonam were instituted at Saint Michael's Medical Center and the Veterans Administration Medical Center. The authors present the encouraging results of their work.

Aztreonam, the first monobactam antibiotic to undergo clinical investigation, is a novel monocyclic β-lactam with a wide aerobic gram-negative bacterial spectrum and high degree of β-lactamase stability. Included in the antimicrobial spectrum of this compound are commonly encountered hospital- and community-acquired gram-negative organisms such as *Pseudomonas aeruginosa*, *Serratia marcescens*, *Enterobacter* sp., indole positive, and negative *Proteus*, *Klebsiella* sp., *Escherichia coli*, *Hemophilus influenzae*, and *Citrobacter freundii*. These organisms frequently are the etiological agents of community-acquired urinary tract infections and nosocomially acquired infection.

The potential of this new compound in treating both community- and hospital-acquired gram-negative aerobic bacillary infection is intriguing, especially as an alternate to the nephrotoxic aminoglycosides and expensive third-generation cephalosporins when dealing with multiply-resistant organisms. Studies of efficacy and safety of aztreonam were instituted at both Saint Michael's Medical Center, Newark, and the Veterans Administration Medical Center, East Orange. Results of those studies are presented in part, including a comparative study of aztreonam versus cefamandole in complicated urinary tract infections, and an open study of aztreonam in difficult gram-negative infections. Both of these studies were preceded by testing of successive gram-negative clinical isolates at both hospitals for susceptibility to aztreonam. Several hundred organisms including many multiply resistant gram-negative rods were screened at both institutions. The encouraging results of those laboratory investigations led to the clinical trials presented here.

METHODS

Hospitalized patients were eligible for entrance into the studies if a clinical diagnosis of infection was bacteriologically confirmed by positive cultures of appropriate specimens. Radiographs were used as supportive evidence in pneumonia. Urine cultures were considered positive when the concentration of the isolated organism was at least 10^5 colony-forming units per ml. Susceptibility studies were done on all isolates by the modified disc method of Kirby and Bauer.

Patients were excluded from the study if they had a history of anaphylactic reaction or type I allergic reaction following administration of a β-lactam antibiotic, received antibiotic therapy within five days prior to the initiation of the study, had hepatic disease prior to the initiation of therapy, had renal insufficiency (creatinine clearance <30 ml/min), were pregnant or nursing, received concomitant antimicrobial therapy (except in the open study where specific gram-positive coverage was allowed), and those in whom a Foley catheter could not be removed within three days of institution of antimicrobial therapy in cases of urinary tract infection.

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Aztreonam, 1 g every eight hours (2 g every eight hours if sepsis was suspected) was given intravenously to patients who were entered into the study. In the comparative study, aztreonam or cefamandole 1 g every eight hours was given intravenously according to a 2:1 randomization schedule. All patients had CBC, platelet count, Coombs' test, serum electrolytes, blood urea nitrogen, creatinine, glutamic-oxalacetic transaminase (SGOT), glutamic-pyruvic transaminase (SGPT), alkaline phosphatase, lactic dehydrogenase, total bilirubin, and urinalysis prior to initiation of

TABLE 1

Characteristics of Patients

	Aztreonam	Cefamandole
No. evaluable/total	19/21	7/10
Average age	57.2 years	67.5 years
Sex (female/male)	18/1	6/1
Anatomical or neurological abnormality of urinary tract	12/19	4/7
Foley catheter	8/19	3/7
Previous urinary tract infection	14/19	5/7
Urosepsis	1/19	1/7
Duration of therapy—days	7.3	7.5
Total dose—g	19.8	22.1

TABLE 2

Bacterial Isolates and KB Zones in Comparative Study

Aztreonam			Cefamandole	
No.	**(Zones)**	**Pathogen**	**No.**	**(Zones)**
8	(29, 18, *28, 29, 39, 36, 36)	*Escherichia coli*	4	(27, 29, 25,25)
5	(15, 32, 24, 26, 25)*	*Pseudomonas aeruginosa*	1	(16)
2	(30.30)	*Klebsiella pneumoniae*	1	(26)
1	(35*)	*Proteus mirabilis*	1	
1	(28)	*Providencia*	0	
0		*Citrobacter freundii*	1	(27)
1	(20)	*Enterobacter cloacae*	0	
1	(12*)	*Acinetobacter anitratus*	0	

*Eight isolates treated with aztreonam were resistant in vitro to cefamandole including five *Pseudomonas aeruginosa.*

TABLE 3

Clinical/Bacteriological Results of Comparative Study

Treatment Group	No. Clinical Cure/No. Evaluable	No. Bacteriological Cure/No. Evaluable	No. Superinfection/ No. Evaluable	No. Relapse/ No. Evaluable	No. Reinfection/ No. Evaluable
Aztreonam					
Last day of therapy	18/19*	18/19*	3/19 (all asymptomatic)	—	—
5 to 9 days post-therapy	17/17	12/17	—	0/17	1/17
Cefamandole					
Last day of therapy	6/7	6/7†	2/7 (all asymptomatic)	—	—
5 to 9 days post-therapy	6/6	3/6	—	1/6	2/6

*Patient lost to followup on last days of therapy (no terminal or 5 to 9 days post-therapy culture).
†One patient persisted with *Pseudomonas* urinary tract infection, later treated successfully with aztreonam.

TABLE 4

Aztreonam: Open Study

No.	Age	Sex	Diagnosis	Underlying Disease	Organism	Response	
						Clinical	Bacteriological
1.	57	M	Recurrent UTI	Paraplegia	*P. mirabilis*	Improved	Reinfected *(P. aeruginosa)*
2.	53	M	Urosepsis	Paraplegia	*P. mirabilis*	Improved	Sterile (Blood and urine)
3.	28	M	Recurrent UTI	Urethral stricture	*K. pneumoniae*	Cured	Sterile
4.	62	M	Complicated UTI	TURP CVA tuberculosis	*K. pneumoniae*	Improved	Reinfected *(P. aeruginosa)*
5.	76	M	Complicated UTI	Multiple sclerosis	*P. mirabilis*	Cured	Recurrence
6.	75	M	Complicated UTI prostatitis	BPH D.M. hypertension	*S. marcescens* *K. pneumoniae*	Cured	Reinfection *(E. coli)*
7.	66	M	Complicated UTI	Carcinoma prostate	*Ent. cloacae*	Cured	Reinfected *(E. coli)*
8.	56	M	Urosepsis	Carcinoma colon	*E. coli*	Cured	Sterile
9.	65	M	Complicated UTI	Quadraplegia Rh. arthritis	*P. mirabilis* *S. marcescens*	Improved	Recurrence *(asymptomatic)*
10.	53	M	Sepsis, Phlebitis	Alcoholism	*Ent. cloacae*	Cure	Cure
11.	51	M	1° Sepsis	Alcoholism hepatitis	*E. coli*	Cure	Cure
12.	59	M	Decubitus cellulitis/ Osteomyelitis	Paraplegia	*P. aeruginosa*	Cure	Reinfected *(P. aeruginosa)*
13.*	63	M	Decubitus acute exacerbation	R. Arthritis	*P. aeruginosa* *P. mirabilis*	Cure	Cure
14.	57	M	Decubitus acute exacerbation	Paraplegia (Failed on Cefoxitin-R)	*Ent. aerogenes*	Cure	Cure
15.	25	M	Decubitus acute exacerbation	Paraplegia	*P. aeruginosa*	Cure	Cure
16.	50	M	Pneumonia	Emphysema (Failed on ampicillin-R)	*H. influenzae*	Cure	Cure

*Received concomitant clindamycin therapy.

therapy. These studies were repeated every five days, at the termination of the study, and when deemed necessary by the investigators.

Followup urine cultures were performed between day two and four of therapy, at the end of therapy, at five to nine days post-therapy and four to six weeks post-therapy for patients with urinary tract infections. Followup cultures of blood, drainage from wounds and sputum were obtained in patients with sepsis, wound infection, and pneumonia when possible.

Clinical response was followed daily; bacteriological cure was defined as elimination or reduction of the original pathogen to less than 10^3 cfu/ml both during and at termination of therapy in urinary tract infection. Relapse, recurrence, reinfection, and superinfection were defined in the usual manner.

RESULTS—COMPARATIVE URINARY TRACT INFECTION STUDY

Thirty-one patients were entered into the comparative urinary tract infection study. Twenty-one patients were assigned to receive aztreonam of which two were excluded for sterile pretherapy urine cultures. Ten patients were randomized to receive cefamandole, 1 patient was excluded because of protocol violation and 2 patients were excluded due to sterile pretherapy urine cultures (Table 1). Bacterial isolates from patients in the comparative study are shown in Table 2.

All isolates that were susceptible to cefamandole also were susceptible to aztreonam, however eight of the isolates susceptible to aztreonam were resistant to cefamandole by modified Kirby Bauer Disc susceptibility testing.

CLINICAL AND BACTERIOLOGICAL RESULTS

Escherichia coli was the most frequent isolate in 12 (49 percent) patients, followed by *Pseudomonas aeruginosa* in 6 (23 percent) patients, and *Klebsiella pneumoniae* in 3 (12 percent) patients. Two of the 26 evaluable patients were bacteremic, with *E. coli*, 1 patient in the aztreonam group and in the cefamandole group. Both of the blood isolates were susceptible to aztreonam and cefamandole and both were cured bacteriologically and clinically (Table 3).

All of the patients in the aztreonam group responded satisfactorily, but one patient did not have culture at termination of therapy (1/19) and two patients did not have followup at five to nine days. However, one patient was reinfected at short-term followup with *Klebsiella pneumoniae* and three patients were superinfected with enterococcus at the termination of therapy. No patients in this group had relapse at short-term followup. The patients with reinfection were all asymptomatic.

One patient treated with cefamandole had persistent infection with a resistant *Pseudomonas aeruginosa*. She was treated successfully with aztreonam after five days of therapy with cefamandole failed to give a clinical/bacteriological response. The cefamandole-treated group had one patient with relapse *(E. coli)* and two patients with reinfection (1 with *Pseudomonas*, 1 with *E. coli*) at the short-term followup. Bacteriological cure at short-term followup therefore was only 50 percent (3/6) for the cefamandole-treated group as compared to 70 percent (12/17) for the aztreonam-treated patients.

RESULTS—OPEN STUDY

Sixteen patients were treated with aztreonam in an open noncomparative trial of efficacy and safety. Table 4 lists the infections and clinical bacteriological response to therapy with aztreonam. Nine patients were treated for complicated urinary tract infections. Five patients were clinically cured and 4 patients showed clinical improvement. Six of the 9 patients treated for urinary tract infections either had recurrence or reinfection on followup and 3 patients had bacteriological cure at the time of followup including 2 patients with urosepsis.

Four patients were treated for sepsis. In addition to the two patients with urosepsis, a patient with intravenous catheter-associated phlebitis/sepsis due to *Enterobacter cloacae* and a patient with primary *E. coli* sepsis were treated successfully with aztreonam.

Four patients were treated with aztreonam for acute exacerbations of chronic decubitus ulcers. All were clinically cured or improved, however one patient showed reinfection with an aztreonam-susceptible *Proteus mirabilis* eight days following elective plastic repair of the decubitus.

An additional patient was treated successfully for a *Hemophilus influenzae* lower respiratory tract infection which had failed to respond to ampicillin. The isolate proved to be resistant to ampicillin and susceptible to aztreonam.

ADVERSE REACTIONS

Adverse drug reactions were limited to one patient treated with aztreonam and consisted of a β-lactam type rash. The patient in which the rash occurred had AIDS and this may have been a factor in the occurrence.

DISCUSSION

Aztreonam performed well in both comparative and open non-randomized trials of efficacy and safety. In vitro results of susceptibility showed aztreonam to have superior activity against the bacterial urinary tract pathogens isolated in the comparative study than did cefamandole. Enterococcal asymptomatic superinfection or reinfection occurred in 4 of 19 patients treated with aztreonam and 2 of 7 patients treated with cefamandole. In the open study, recurrences and reinfection after therapy with aztreonam were frequent (7 of 16 patients treated). This is not surprising given the underlying disorders in the patient population studied with complicated UTI and decubitae.

CONCLUSION

Aztreonam appears to offer effective and specific aerobic gram-negative therapy in moderate to severe complicated infections.

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AZTREONAM: WORLDWIDE CLINICAL EXPERIENCE WITH THE FIRST MONOBACTAM

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Aztreonam, a unique β-lactam antibiotic, was produced and developed by scientists at The Squibb Institute for Medical Research in Princeton from a precursor which they discovered in the Pine Barrens of New Jersey.

Aztreonam (AZACTAM® for Injection, Squibb) is the first member of a new and unique class of β-lactam compounds designated by researchers at The Squibb Institute for Medical Research as monobactams (monocyclic bacterially produced β-lactam antibiotics). Unlike other naturally occurring β-lactam antibiotics, which are produced by fungi or streptomycetes, the monobactams are produced by bacteria. The first such compound was SQ 26,180, a naturally occurring substance produced by *Chromobacterium violaceum.* The monobactams are distinguished from other β-lactam antibiotics, e.g. penicillins and cephalosporins, by having a single ring structure. The isolation of naturally occurring monobactams led to the synthesis of aztreonam, which was found to possess several properties making it ideal for clinical development.

The uniqueness of the monocyclic ring itself, together with the design of the aztreonam molecule which confers specific antibacterial properties, represents a significant advance in antibiotic development.

Targeted Spectrum. Aztreonam is targeted specifically against the aerobic gram-negative bacteria, including multiply drug-resistant strains, which have become a common cause of nosocomial infection. Its spectrum encompasses the majority of members of the *Enterobacteriaceae*, as well as *Pseudomonas aeruginosa, Hemophilus influenzae*, and *Neisseria gonorrhoeae.* The exceptional activity of aztreonam against these organisms appears to be a function of its high and predominant affinity for penicillin-binding protein 3 (PBP 3). The anti-gram-negative bacterial activity of aztreonam in vitro generally is equal to or exceeds that of the third-generation cephalosporins and aminoglycosides.

Resistance to Enzymatic Degradation. Aztreonam has a high degree of stability to both plasmid- and chromosomally mediated β-lactamases, equal or superior to that of the third-generation cephalosporins. Aztreonam also is stable to the β-lactamase produced by penicillin-resistant *Staphylococcus aureus;* this may be relevant in the treatment of mixed pathogen infection, when both gram-positive and gram-negative organisms are present. As expected, aztreonam is highly stable to the aminoglycoside-deactivating enzymes that inactivate antibiotics such as gentamicin, tobramycin, and amikacin.

Lack of β-Lactamase Induction. Unlike antibiotics such as cefoxitin, cefotaxime, and ceftazidime, aztreonam does not induce in vitro the chromosomally mediated cephalosporinases in such strains as *Enterobacter, Serratia, Providencia, Proteus,* and *Pseudomonas.*

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Potential for Preservation of Colonization Resistance. Aztreonam has minimal activity against gram-positive bacteria and anaerobes. In vivo experiments with hamsters have demonstrated a consequent lack of disturbance of normal cecal anaerobic flora (obviating *C. difficile* overgrowth with concomitant toxin production).

Low Immunogenicity. In vitro studies demonstrated a lack of significant cross-reactivity between aztreonam and IgE antibody to benzylpenicillin, in contrast to the high degree of cross-reactivity between cephalothin and penicillin antibody.[1]

When combined in vitro with other β-lactam antibiotics and aminoglycosides, aztreonam usually has an additive (and often synergistic) effect against gram-negative aerobic organisms. Also, there is no antagonism to the activity of broad-spectrum antibiotics against gram-positive organisms. Therefore, it appeared that aztreonam could be used concurrently with complementary antibiotics to provide broad-spectrum coverage in empiric therapy situations. For the above reasons, following standard preclinical, pharmacologic,[2] and toxicologic[3] testing, the clinical pharmacology program for aztreonam was started in the spring of 1981.

CLINICAL PHARMACOLOGY

Aztreonam was introduced into human volunteers in April 1981, at the Clinical Pharmacology Unit of The Medical Center at Princeton. The results of this first pharmacokinetic study were presented at a symposium held at the XII International Congress of Chemotherapy in Florence, Italy, in July 1981.[4] This pivotal study revealed that intravenously administered aztreonam was well-tolerated and achieved potentially therapeutic concentrations in the serum and urine. Key pharmacokinetic properties of aztreonam in healthy volunteers were delineated in a number of early pharmacology studies and are summarized in Table 1.

TABLE 1

Pharmacokinetic Properties of Aztreonam[30]

	Aztreonam
Protein Binding, %	
Healthy volunteers	56
Patients	30
Half-life, hours	1.7
Volume of distribution, liters	11
Impaired elimination with renal dysfunction	Yes
Urinary excretion in 24 hours, %	68
Biliary excretion, %	15

Aztreonam administered parenterally is eliminated primarily in the urine in unchanged form, and also undergoes biliary excretion, estimated for healthy subjects to be 15 percent, based upon fecal recovery of radioactivity after a 0.5-g parenteral dose.[5] The elimination of aztreonam is impaired in the presence of renal failure, such that the half-life is about three times normal and the serum clearance is one-fourth normal.[6] Aztreonam undergoes metabolism to a minor extent, as indicated by the appearance of 7 percent of a 0.5-g dose in the urine as the open-β-lactam-ring derivative, SQ 26,992 (analogous to the penicilloate metabolite of penicillin).

Peak serum concentrations of aztreonam administered as 0.5-, 1-, and 2-g 30-minute intravenous infusions to healthy volunteers were 66, 164, and 225 μg/ml, respectively.[7] In comparison, serum values at 8 hours after dosing were 1.8, 3.0, and 8.5 μg/ml, respectively, and at 12 hours were 0.2, 0.9, and 1.9 μg/ml, respectively. Urinary levels after 30-minute infusions of 0.5-, 1-, and 2-g doses were 1000, 3200, and 5700 μg/ml during 0 to 2 hours and were 23, 52, and 110 μg/ml during 8 to 12 hours after dosing, respectively.[7] The serum levels of aztreonam following single 0.5-, 1-, and 2-g doses exceed the MIC_{90} for *Neisseria* sp, *H. influenzae,* and most genera of the *Enterobacteriaceae* for 8 hours (for *Enterobacter* sp the 8 hour serum levels exceed the MIC_{90} for 80 percent of strains). For *Pseudomonas aeruginosa,* a single 2-g intravenous dose produces serum levels that exceed the MIC_{90} for 4 to 6 hours. All of these doses of aztreonam result in average urine levels exceeding the MIC_{90} for the same pathogens for up to 12 hours.

Probenecid does not have a major effect on the pharmacokinetic profile of aztreonam, although elimination of aztreonam by net tubular secretion is reduced.[8] No clinically significant drug interaction has been found between aztreonam and cephradine, clindamycin, gentamicin, metronidazole, or nafcillin.[9] Aztreonam undergoes clearance by both hemodialysis and chronic ambulatory peritoneal dialysis (CAPD); hemodialysis can remove about 50 percent of a dose in 4 hours, while CAPD removes only 10 percent of a dose in 48 hours.[10,11]

The pharmacokinetics of aztreonam in pediatric patients have been reported.[12] Newborns less than 7 days old and less than 2500 g body weight had delayed elimination of aztreonam (half-life 5.7 hours) compared to adults. An in vitro study showed that, under physiologic conditions, aztreonam did not displace bilirubin bound to albumin, whereas sulfisoxazole, moxalactam, and cefoperazone did increase unbound bilirubin.[13]

Aztreonam appears to be only poorly immunogenic when given in multiple doses to humans[1] and to be poorly cross-reactive in vivo[14] and in vitro[1] with IgE antibody to penicillin. Also, multiple intravenous doses of aztreonam produce marked reduction in the numbers of fecal aerobic gram-negative bacilli without notably altering the numbers of fecal anaerobes.[15]

The favorable pharmacokinetic profile of aztreonam served as the basis for the clinical trial program, which was initiated at the end of 1981. This program was designed to evaluate aztreonam in comparative and noncomparative studies.

CLINICAL RESULTS*

Worldwide clinical trials, encompassing both open (noncomparative) and comparative studies, were designed to evaluate the safety and efficacy of aztreonam in the treatment of a wide variety of infections due to susceptible aerobic gram-negative organisms. To date, data from a total of 3,706 aztreonam-treated patients (346 having been entered into single-dose studies and 3,360 in multiple-dose studies) have been analyzed. Of these, 1,980 met predetermined criteria[16,17] for an evaluation of clinical and microbiologic efficacy; a total of 543 patients treated with control antibiotics met these same criteria.

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*Other publications have presented the data and results from the ongoing clinical trials.[16-23]

These clinical trials were designed to evaluate the therapeutic efficacy of aztreonam as a single 1-g intramuscular (IM) dose for acute uncomplicated gonorrhea and acute, uncomplicated cystitis and as multiple dose therapy (0.5 to 2 g IM or IV every 6, 8, or 12 hours, most commonly 1 g every 8 hours) for the treatment of more serious infections including those involving the urinary and lower respiratory tracts, blood, bones and joints, skin and soft tissues, intra-abdominal and female genital tissues.

Patients with acute, uncomplicated gonorrhea and acute, uncomplicated cystitis were treated in randomized comparative studies in which the respective control drugs were spectinomycin (single 2 g IM dose) and amoxicillin (250 mg q.i.d. given orally for ten days).[16] Open multiple-dose studies encompassed all of the above types of infections; patients with urinary tract, lower respiratory tract, intra-abdominal, and gynecologic infections also were evaluated in randomized comparative trials in which the control antibiotics were cefamandole (urinary tract infections), tobramycin (lower respiratory tract, and intra-abdominal infections), and gentamicin (gynecologic infections).[16]

Generally excluded from the studies were patients with a history of allergy to the study drugs or immediate hypersensitivity reactions to penicillin, neutropenia, i.e. neutrophil count <1000/mm^3, pregnant or lactating women, or concurrent illness that in the opinion of the investigator would make the patient unsuitable for enrollment.

In the multiple-dose studies, a minimum of five days of therapy was required to establish cure. Patients who were treated for less than five days could be assessed as therapeutic failures provided a minimum of 48 hours of treatment was given.

Antibiotics with a spectrum of activity limited to gram-positive and/or anaerobic organisms, most commonly clindamycin, were administered concurrently for "mixed" pathogen infections. The dosage for concurrently administered antibiotics as well as the control drugs was based on the manufacturers' recommendations. Thus, therapeutic regimens could be modified to provide broad spectrum therapy of known or suspected "mixed" pathogen infections or utilize aztreonam alone for treatment of those infections in which aerobic gram-negative organisms were the only causative pathogens.

The microbiologic and clinical responses for all evaluable patients are shown in Table 2. The favorable clinical response rates were all 91 percent or above while the microbiologic cure rates ranged from 80 percent for lower respiratory tract infection to 99 percent for gynecologic infection.

In cystitis studies, favorable clinical responses and microbiologic cures were achieved in 93 percent and 84 percent of 56 patients, respectively, who received a single dose of aztreonam and in 96 percent and 93 percent of 45 patients, respectively, who were treated with a ten-day course of amoxicillin. *Escherichia coli* was the causative organism in 90 percent of infections in patients in both treatment regimens.

Excellent microbiologic response rates were achieved with single 1-g doses of aztreonam when used in the treatment of acute uncomplicated gonorrhea. Ninety-five percent of 128 men and 100 percent of 81 women given aztreonam were microbiologically cured. Comparable microbiologic cure rates were achieved with spectinomycin; 97 percent of 111 men and 96 percent of 90 women responded. The overall microbiologic cure rate was 97 percent for both treatment regimens. Penicillinase-producing strains of *N. gonorrhoeae* were responsible for 14 percent of the infections in the aztreonam group and 13 percent of those given spectinomycin.

Overall, 1,461 (85 percent) of 1,715 evaluable pa-

TABLE 2

Overall Clinical and Microbiologic Responses

Diagnosis	**Patients (N)**	**Favorable Clinical Response (%)**	**Microbiologic Cure (%)**
Single dose:			
Acute gonorrhea	209[1]	94	97
Acute cystitis	56	93	84
Multiple dose:			
Urinary tract infection	712[2]	99	83
Skin infection	317[3]	96	86
Lower respiratory tract infection	348[3]	91	80
Gynecologic infection	73	99	99
Intra-abdominal infection	121[4]	91	88
Septicemia	122[2]	95	98
Bone/joint infection	22	100	91

No clinical response information available for: [1](19 pts.); [2](2 pts.); [3](3 pts.); [4](1 pt.).

TABLE 3

*Microbiologic Cure Rates**
Multiple-Dose Studies

Enterobacteriaceae	**UTI**	**LRTI**	**Intra-abd**	**Gyn**	**Skin**	**Bone**	**Septicemia**	**Overall Total T/C***	
E. coli	324/278	27/25	40/38	27/26	23/18	3/2	54/53	498/440	(88%)
Klebsiella sp	63/56	44/42	8/7	8/8	12/12	—	14/14	149/139	(93%)
Enterobacter sp	36/31	23/23	5/5	1/1	28/22	3/2	12/12	108/96	(89%)
Serratia sp	34/30	15/11	1/1	—	19/19	2/2	8/8	79/71	(90%)
Proteus sp	67/63	8/6	3/3	6/6	37/36	2/2	7/7	130/123	(95%)
Hemophilus sp	—	54/54	—	—	2/2	—	4/4	61/61	(100%)
Other	16/14	20/17	10/10	14/14^{+}	9/9	1/1	8/8	77/72	(94%)
Pseudomonads	127/92	93/53	13/11	—	94/77	7/7	7/6	341/246	(72%)
Multiple organisms	45/29	64/46	41/31	17/17	93/79	4/4	8/7	272/213	(78%)
Total	712/593 (83%)	348/277 (80%)	121/106 (88%)	73/72 (99%)	317/274 (86%)	22/20 (91%)	122/119 (98%)	1715/1461	(85%)

*T/C = Total number patients/patients cured.
$^{+}$ = All 14 patients had infection due to *N. gonorrhoeae.*

TABLE 4

Clinical Adverse Effects (ADEs)
Aztreonam Multiple-Dose Studies (3,360 patients)

Reaction	**Number of Patients (% of Patients Treated)**
Dermatologic (1.8%)	
Rash	44 (1.3%)
Rash with eosinophilia	10 (0.3%)
Pruritus	5 (0.1%)
Purpura	3 (0.1%)
Gastrointestinal (2.0%)	
Diarrhea	26 (0.8%)
Nausea/vomiting	19 (0.6%)
Taste alteration	8 (0.2%)
Colitis or *C. difficile* diarrhea	6 (0.2%)
Jaundice/hepatitis	4 (0.1%)
Oral lesions	4 (0.1%)
Local Reactions (1.7%)	
Thrombophlebitis/phlebitis	41 (1.5%)*
Discomfort/swelling at injection site	15 (2.6%)**
CNS-Related (0.4%)	
Headache	6 (0.2%)
Dizziness	2 (0.1%)
Other	5 (0.1%)
Miscellaneous (0.9%)	
Fever/chills/sweating	6 (0.2%)
Vaginitis	5 (0.1%)
Fatigue	4 (0.1%)
Hypersensitivity reaction	2 (<0.1%)
Bleeding	2 (<0.1%)
Other	12 (0.4%)
Total Number of ADEs	229
Total Number of Patients Experiencing ADEs	209 (6.2%)

*Based upon the 2,775 patients who received aztreonam intravenously.
**Based upon the 585 patients who received aztreonam intramuscularly.

tients treated with aztreonam in multiple-dose studies were microbiologically cured. As shown in Table 3, the overall microbiologic cure rates of these infections ranged from 72 percent for *Pseudomonas* to 100 percent for other common aerobic gram-negative pathogens.

In comparative and noncomparative multiple-dose studies, the incidence of reinfection, i.e. infection following therapy due to a new organism, ranged from 3 percent in lower respiratory tract infection to 7 percent in urinary tract infection, while the incidence of microbiologic relapse, i.e. recurrence of infection after therapy due to the original pathogen, was approximately 5 percent; the incidence of reinfection and relapse was comparable to that found with the control drugs.

In single 1-g intramuscular dose trials of aztreonam for the treatment of acute, uncomplicated gonorrhea or acute, uncomplicated cystitis, 6 of 346 patients (1.7 percent) experienced clinical adverse effects reported as being related or possibly related to aztreonam (ADEs), most commonly nausea and/or vomiting. No serious ADEs were reported. Generally, aztreonam injections were well-tolerated in these patients.

A summary of ADEs reported in the 3,360 patients entered into multiple-dose studies is shown in Table 4. The most common ADEs among all patients treated with aztreonam were local reactions at the injection site, including phlebitis or thrombophlebitis, rash or other dermatologic finding, diarrhea, and nausea and/or vomiting.

Diarrhea, classified by the investigators as related or possibly related to aztreonam, usually was not severe. However, five patients developed diarrhea with stools positive for *Clostridium difficile* or its toxin, and one developed culture-negative pseudomembranous colitis. Potentially confounding factors, such as prior or concurrent treatment with other antibiotics, prior or concurrent therapy with cytotoxic or immunosuppressive agents, or the presence of diarrhea at the outset of aztreonam therapy, were evident in most of these patients. Aztreonam previously has been shown not to induce colitis in the well-established hamster model, and the antibiotic has little effect on normal human anaerobic intestinal flora.[24,25]

Four patients developed hepatic abnormalities which were reported as ADEs. One case of hepatitis, believed to be related to aztreonam, was manifested only by elevations in serum transaminase and lactic dehydrogenase values. These values returned toward normal following discontinuation of drug. Two cases of jaundice were reported to be possibly due to aztreonam therapy. In both patients total serum bilirubin levels were greater than 1.5 mg/dl prior to receiving the antibiotic. The fourth patient developed elevated serum transaminase values (hepatitis, possibly aztreonam-related) on the fifth day of treatment. The SGOT and SGPT levels already had decreased when aztreonam therapy was stopped the following day, and by the time of the last available evaluation these values had returned to near normal levels.

Two seriously ill patients developed significant bleeding, reported as ADEs, while receiving aztreonam and other medications; in neither case could the bleeding (due to thrombocytopenia in one patient and of uncertain etiology in the other) be attributed definitely to aztreonam. Alteration of prothrombin times occurred rarely in patients given aztreonam. No patients were reported as having evidence of clinical bleeding due to aztreonam-induced hypoprothrombinemia.

Aztreonam was well-tolerated and effective against a wide variety of infections.

Combining results in multiple-dose studies of lower respiratory tract infections and urinary tract infections, abnormalities in serum creatinine ($\geqq$0.5 mg/dl increase if pretreatment value <3.0 mg/dl, $\geqq$1.0 mg/dl increase if pretreatment value $\geqq$3.0 mg/dl) were observed in 15 percent of those patients who received aminoglycosides and in 5 percent of those who received aztreonam. When laboratory abnormalities occurring one week or more post-therapy were excluded, the incidence in these aminoglycoside groups decreased to 9 percent. In comparative trials, there were no significant differences in the incidence of patients with increases in SGOT or SGPT (>3 times upper normal limits or >4 times pretreatment if no normal limits available) when aztreonam was compared to control antibiotic. In two of nine trials statistically greater increases in the mean values of SGOT and/or SGPT over the course of therapy were observed in those administered aztreonam compared to those given control agents. However, these differences (in the range of 4 to 6 I.U.) were of a low order of magnitude and, by themselves, were not considered clinically significant.

Two patients manifested ADEs classified as hypersensitivity reactions. One, reportedly penicillin-sensitive by skin test, developed fever, rash, and hypotension on day 11 of therapy, with reappearance of an allergic-type response when challenged with aztreonam 5 days later. The other, with a history of rash when administered penicillin or nitrofurantoin, manifested urticaria and pharyngeal edema on day 3 of aztreonam treatment. However, a lack of interaction between aztreonam and IgE antibody to penicillin has been demonstrated in studies designed specifically to investigate potential cross-reactivity.[26]

Only 79 of all 3,360 patients (2.4 percent) treated with aztreonam had therapy discontinued because of an ADE or abnormal laboratory test result. Rash was the single most common reason cited for stopping therapy.

In the comparative studies, suprainfections (new infections occurring during treatment at the original site of infection and which themselves were treated) were reported in 1 to 7 percent of aztreonam-treated patients and 0 to 6 percent of control patients, with the incidence varying by treatment indication. The most common suprainfecting organisms in patients treated with aztreonam were group D streptococci (including enterococci), staphylococci, *Streptococcus pneumoniae*, and *Acinetobacter calcoaceticus*; in patients treated with control antibiotics, *Pseudomonas aeruginosa*, staphylococci, and *Escherichia coli* most frequently were found. Underlying host factors, such as chronic respiratory disease, advanced age, and urethral catheterization, may play an important role in the development of these types of secondary bacterial infections.[27-29]

These data indicate that aztreonam is a well-tolerated antibiotic whose safety profile is comparable to that of other β-lactam compounds.

DISCUSSION

First-generation cephalosporins show varying degrees of susceptibility to β-lactamases, but they lack high intrinsic activity against most gram-negative organisms and none is active against *Pseudomonas aeruginosa*. On the other hand, the aminoglycosides, although ototoxic and nephrotoxic, demonstrate high intrinsic activity against a large percentage of aerobic gram-negative pathogens, including *Pseudomonas aeruginosa*. Second-generation cephalosporins show increased stability to gram-negative β-lactamases compared to earlier cephalosporins, but they lack high intrinsic activity against the majority of gram-negative organisms and have essentially no activity against *Pseudomonas*. The third-generation cephalosporins include, among others, ceftizoxime, cefotaxime, ceftazidime, moxalactam, and cefoperazone. Usually these compounds are highly active against the majority of aerobic gram-negative organisms and are resistant to hydrolysis by β-lactamases, but they have variable activity against *Pseudomonas aeruginosa*.

The recent trend in antibiotic research has been toward the development of potent broad-spectrum compounds for use as empiric therapy. An unwanted consequence of introducing such "broad-spectrum" antibiotics has been an increase in the incidence of serious side effects such as bleeding and colitis, as well as an increase in the incidence of nosocomial infections and a loss of therapeutic flexibility. Our research efforts were directed toward finding an antibiotic that could be used for specific therapy against infections due to aerobic gram-negative pathogens without harming normal bacterial flora. These efforts culminated in the development of aztreonam, the first of a new class of antimicrobial agents, the monobactams.

The unique molecular structure of aztreonam results in a particularly high degree of resistance to β-lactamases (a major mechanism of bacterial resistance to penicillins and cephalosporins), exceptional activity against aerobic gram-negative pathogens, and little or no activity against gram-positive organisms and anaerobes.

Preclinical and clinical data demonstrated that aztreonam has marked activity against a wide range of aerobic gram-negative pathogens (β-lactamase-producing and nonproducing). It offers the clinician an opportunity for initial empiric antimicrobial therapy as either a single agent or an agent to be used concurrently with one that has activity against anaerobes and gram-positive aerobes, thereby achieving broad-spectrum coverage pending microbiologic identification of the pathogen or pathogens. Once the causative pathogens are identified and antibiotic susceptibilities determined, aztreonam may be used as the sole antibiotic therapy to treat infections due to aerobic gram-negative organisms, or may be used in combination with antibiotics that have activity against gram-positive and/or anaerobic organisms to treat "mixed" pathogen infections. This flexibility of therapy allows the clinician to modify the therapeutic regimen to suit the type of infection being treated. Subsequent therapy, then, can be guided in a more specific fashion in lieu of continued and unnecessary administration of broad-spectrum β-lactam antibiotics which may disturb the normal intestinal flora and produce detrimental side effects. In addition, combination therapy may be specifically targeted against certain organisms. Results from microbiologic tests and animal studies demonstrated a potential synergy when combining aztreonam with aminoglycosides. This may be of value, for example, in the treatment of life-threatening infections with *Pseudomonas*, such as may occur in patients with septicemia, cystic fibrosis, and granulocytopenia.

CONCLUSION

Aztreonam was shown to be effective for parenteral use in patients with infections of the urinary tract, lower respiratory tract, skin and skin-structures, peritoneum and adjacent structures, and bone and joint tissues, as well as in patients with septicemia and gynecologic disease, when these infections are due to susceptible organisms which include most genera of the *Enterobacteriaceae*, as well as *Pseudomonas* sp (including *P. aeruginosa*), *Neisseria gonorrhoeae*, and *Hemophilus influenzae*.

Except for patients with acute, uncomplicated cystitis or gonorrhea, for whom single 1-g doses are adequate, adults require doses ranging from 0.5 to 2 g, administered every 6 to 12 hours either intravenously or intramuscularly; the exact dosage and the route of administration depend on the nature and severity of the infection. For mixed infections, or for empiric therapy when the causative organisms have not yet been identified, aztreonam may be administered concurrently with appropriate antibiotics having activity against gram-positive and anaerobic organisms.

The side effect profile of AZACTAM® is qualitatively similar to other β-lactam agents, i.e. local reactions at the injection site, rash, occasional increases in serum transaminase values, and minor gastrointestinal disturbances. Approximately 2 percent of patients had therapy discontinued because of side effects, most commonly due to the occurrence of rash.

Aztreonam has several attributes, among which are organism specificity which permits preservation of colonization resistance, resistance to β-lactamase enzymes, lack of induction of bacterial β-lactamases, and apparent lack of cross-allergenicity with penicillin. In the clinic, aztreonam was well-tolerated and effective against a wide variety of infections due to a broad spectrum of susceptible aerobic gram-negative pathogens.

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AZTREONAM: THERAPY FOR INFECTIONS IN CANCER PATIENTS

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Aztreonam is a new monocyclic β-lactam antibiotic with broad-spectrum activity against gram-negative bacilli. In a prospective randomized trial in neutropenic cancer patients, aztreonam alone or in combination with amikacin was effective against gram-negative infections.

Substantial advances have been made in our understanding of the infectious complications of cancer patients during the past two decades. Nevertheless, infection remains a major cause of morbidity and mortality in these patients. Although there has been a resurgence of gram-positive infections in these patients in recent years, gram-negative bacilli still are responsible for the majority of infections. Aminoglycosides have been incorporated into most antibiotic regimens used for the treatment of infection in these patients because their spectrum of activity includes most gram-negative bacilli. Unfortunately, several problems have emerged with the routine use of these antibiotics in cancer patients. Many cancer patients are neutropenic when they develop infection, and aminoglycosides often are ineffective in neutropenic patients. These patients have repeated infections and often require prolonged antibiotic therapy, both factors which increase the likelihood of aminoglycoside-induced nephrotoxicity. Furthermore, cancer patients may be treated with other nephrotoxic agents, such as Platinol®, which enhances the potential for nephrotoxicity during aminoglycoside therapy. Consequently, new antibiotics with broad-spectrum activity against gram-negative bacilli are of special interest.

AZTREONAM IN PATIENTS WITH CANCER

Aztreonam is a new monocyclic β-lactam antibiotic that is active against most gram-negative bacilli, but is relatively inactive against gram-positive bacteria and anaerobes.[1] We tested the in vitro activity of this antibiotic against clinical isolates of gram-negative bacilli cultured from blood specimens of cancer patients.[2] The results of this study are summarized in Table 1. Aztreonam was very active against most of the aerobic gram-negative bacilli. A concentration of 3.12 μg/ml inhibited 98 percent of the isolates of *Escherichia coli*, 100 percent of *Klebsiella pneumoniae*, *Serratia marcescens*, and *Proteus* sp., 90 percent of *Citrobacter* sp., 77 percent of *Pseudomonas aeruginosa*, 64 percent of *Enterobacter* sp., but only 28 percent of *Acinetobacter calcoaceticus*. We compared the activity of aztreonam with the activity of the aminoglycoside, tobramycin. Aztreonam was more active than tobramycin against *E. coli*, *K. pneumoniae*, *Proteus* sp., and *Citrobacter* sp. However, tobramycin was more active than aztreonam against *Enterobacter* sp., *P. aeruginosa*, and *A. calcoaceticus*.

PHARMACOKINETIC STUDIES IN HEMATOLOGICAL MALIGNANCIES

These encouraging results led us to conduct pharmacokinetic studies in 25 adult patients with hematological malignancies.[3] Aztreonam was substituted for the usual antimicrobial prophylaxis administered to 18 patients entering a laminar air flow unit. These patients were given 1 or 2 g of aztreonam every 8 hours for seven or eight days. The mean peak serum concentration following the administration of 1 g aztreonam over ½ hour intravenously was 75.5 μg/ml and the serum half-life was 1.7 hour. At 6 hours, the mean serum concentration was 5.4 μg/ml. There were no substantial differences in the mean serum concentrations after three to five and six to nine days of aztreonam administration. Following a 2 g dose administered over ½ hour intravenously, the mean peak serum concentration was 177.2 μg/ml and the serum half-life was 2.0 hour. Approximately 60 percent of the drug was excreted in the urine within 8 hours. Small amounts of the drugs were converted to an inactive metabolite, SQ 26,992. With repeated dosing, there was evidence of cumulation of this metabolite in the serum and urine.

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Several studies conducted at this institution have indicated that it is important to maintain continuously adequate serum concentrations of antibiotics in neutropenic patients to obtain maximum therapeutic efficacy.[4,5] Consequently, aztreonam was administered in the following manner as therapy in neutropenic patients. A loading dose of 1.5 g was given over ½ hour, followed by 1.5 g given as a 2-hour continuous infusion every 4 hours thereafter. Pharmacokinetic studies were conducted in seven patients on the second or third day of therapy. The mean peak serum concentration at the end of an infusion was 68.5 μg/ml and it dropped to 29.9 μg/ml at the end of 4 hours (just prior to the next infusion).

The impact of aztreonam administration on the stool flora was determined in the 18 patients who received this antibiotic for one week as prophylaxis while in a laminar air flow unit. Semiquantitative throat and stool specimens were collected before onset of drug administration, on days three to five and again on days seven to nine of drug administration.[6] Aztreonam had little effect on predominant gram-positive throat flora of these patients. Its effect on aerobic and facultative gram-negative bacilli in the feces was profound with only 1 of 48 isolates that were cultured prior to drug administration persisting on days seven to nine. Aerobic gram-positive organisms in the stool were less affected by aztreonam and its effect on anaerobes was variable. Among patients who received 1 g aztreonam every eight hours, the concentration of aerobic gram-negative bacilli decreased from approximately 10^6 to 10^1 per gram stool, whereas the concentration of anaerobes and gram-positive cocci decreased by about 1 log or actually increased during drug administration. The impact of these changes in stool flora on the acquisition of nosocomial organisms could not be assessed because the patients were located in laminar air flow units.

TABLE 1

In Vitro Activity Of Aztreonam

Organism	Isolates	MIC_{50}	MIC_{90}
E. coli	100	0.10	0.20
K. pneumoniae	100	0.05	0.20
S. marcescens	100	0.10	0.39
P. aeruginosa	100	3.12	12.5
Enterobacter sp	72	0.39	50
Proteus sp	29	0.05	1.56
A. calcoaceticus	25	25	50
Citrobacter sp	22	0.22	0.39

$MIC_{50(90)}$ = concentration in μg/ml required to inhibit 50% (90%) of isolates.

AZTREONAM IN NEUTROPENIC PATIENTS: METHOD AND RESULT

Because of its activity against gram-negative bacilli, aztreonam was evaluated in a prospective randomized trial as initial therapy for empiric therapy of fever presumed to be due to infection in neutropenic patients.[7] Patients were randomly assigned to receive: a) aztreonam plus vancomycin (AV); b) aztreonam plus vancomycin plus amikacin (AVA); or c) ticarcillin plus moxalactam (MT). The latter regimen was selected for comparison based on our previous study in which it was as effective as moxalactam plus tobramycin.[8] Vancomycin was included with aztreonam to provide coverage against gram-positive organisms. The objectives of this study were to evaluate the efficacy of aztreonam against gram-negative bacillary infections and to determine whether the addition of amikacin enhanced the activity of aztreonam.

Aztreonam was administered by continuous infusion, as described above. Vancomycin was given intravenously at a dose of 500 mg every 6 hours. Amikacin was given as a loading dose of 150 mg/M^2 over ½ hour followed by 600 mg/M^2/day given by continuous infusion. Patients assigned to the third regimen received ticarcillin 4 g over 2 hours every 4 hours, alternating with moxalactam 2 g over 2 hours every 4 hours. Appropriate cultures and laboratory examinations were performed before and during therapy.[7] The criteria for response to therapy also have been reported elsewhere.[7] Patients responding to therapy were treated for a minimum of seven days, or four days following disappearance of signs of infection, whichever was longer.

A total of 295 febrile episodes have been evaluated in this ongoing study. An additional 52 episodes were excluded because of major protocol violations, death within 24 hours, or proved viral or fungal infection. The three groups of patients were comparable with respect to underlying malignant disease although the proportions of patients with severe neutropenia (<100 neutrophils/mm^3) differed substantially (AV 64 percent, AVA 58 percent, and MT 70 percent; P = .09).

The overall results of this study are presented in Table 2. The 80 percent response rate for AVA was better than the 68 percent response rate for AV (P = .07) and the 61 percent response rate for MT (P = .005). No evidence of infection could be determined during 42 percent of the febrile episodes. AVA produced the highest response rate among those patients with fever of unknown origin (FUO). AV and AVA produced similar response rates in patients with FUO and

TABLE 2

Overall Efficacy of Antibiotic Regimens

	AV		AVA		MT	
	Episodes	**% Response**	**Episodes**	**% Response**	**Episodes**	**% Response**
Total	100	68	97	80	98	61
FUO (Fever of unknown origin)	36	67	41	80	48	67
Documented infection	64	69	56	80	50	56
Organism identified	33	88	28	93	28	64
Organism not identified	31	48	28	68	22	45

TABLE 3

Results by Site of Infection

	AV		AVA		MT	
Infection	**Episodes**	**% Response**	**Episodes**	**% Response**	**Episodes**	**% Response**
Septicemia	14	86	18	94	15	73
Pneumonia	24	42	12	42	13	23
Urinary tract	7	100	5	100	2	100
Soft tissue	10	80	14	100	11	45
Other	9	78	7	57	9	78

TABLE 4

Response to Specific Pathogens

	AV		AVA		MT	
Organism	**Episodes**	**% Response**	**Episodes**	**% Response**	**Episodes**	**% Response**
Total gram-positive	10	9 (90%)	5	4 (80%)	14	6 (43%)
S. aureus	6	5	2	1	4	0
S. epidermidis	2	2	1	1	3	1
Others	2	2	2	2	7	5
Total gram-negative	17	15 (88%)	17	17 (100%)	5	5 (100%)
E. coli	6	6	8	8	2	2
Klebsiella	2	2	2	2	1	1
P. aeruginosa	6	4	5	5	2	—
Others	3	3	2	2	—	—
Polymicrobial	6	5 (83%)	6	5 (83%)	9	7 (78%)
Total	33	29 (88%)	28	26 (93%)	9	18 (64%)

documented infections whereas MT was more effective in patients with FUO. AVA significantly was better than MT among patients with documented infections (80 percent *v* 56 percent; *P* = .013). The superiority of AVA over MT was observed whether or not the infecting organism could be determined. AVA also produced a higher response rate than AV among patients with documented infections (*P* = .21). However, this difference was accounted for by those infections in which the infecting organism could not be determined.

The results related to the site of infection are shown in Table 3. AV and AVA consistently were superior to MT for all sites of infection except the urinary tract. AV and AVA produced similar results against all types of infection except those involving soft tissues in which AVA was superior (80 percent *v* 100 percent *P* = .32). Unfortunately, the randomization process resulted in a maldistribution of the sites of infection. There were twice as many episodes of pneumonia assigned to AV than to the other two regimens and fewer urinary tract infections were assigned to MT. Since the response rates for pneumonias were substantially poorer than other sites and there was no difference in the efficacy of AV and AVA in pneumonias, the overall difference between AV and AVA in documented infections could be due solely to this maldistribution of episodes of pneumonia. The response rates were determined for AV and AVA for all documented infections except pneumonia and were 85 percent and 89 percent, respectively. Hence, the apparent superiority of AVA over AV against documented infections primarily was due to the higher number of episodes of pneumonia assigned to AV. Correcting for the maldistribution of cases of urinary tract infections did not alter

TABLE 5

Response to Therapy According to Trend in Granulocyte Count

Initial Neutrophil Count/mm³	Trend	AV Episodes	AV % Response	AVA Episodes	AVA % Response	MT Episodes	MT % Response
<100	Total	40	63	33	76	34	62
<100	Increased	24	83	21	81	13	85
<100	Unchanged	16	31	12	67	21	48
101-1000	Total	19	79	19	84	10	50
>1000	Total	4	75	4	100	6	50
All	Decreased or unchanged	28	46	22	73	29	52
All	Increased	35	86	34	85	20	65

the poorer results obtained with MT.

Despite the entry of 295 episodes on this study, only 89 (30 percent) were documented infections in which the infecting organism could be identified (Table 4). Hence, the number of infections caused by any single organism was too small to permit any meaningful comparisons of efficacy between these three regimens. However, the two regimens containing vancomycin were more effective than MT against gram-positive cocci (90 percent *v* 80 percent *v* 43 percent; $P = .04$). The only failures with vancomycin were infections caused by *Staphylococcus aureus* and none of the infections caused by this organism responded to MT. The only gram-negative infections that failed to respond were two episodes of *Pseudomonas* infection that did not respond to AV. Overall, the addition of amikacin to AV did not substantially improve the results for gram-negative bacillary infections.

Table 5 presents the results related to the patients' initial neutrophil counts and changes during therapy. Even among patients with severe neutropenia, the response rates were encouraging. The poorest response rates were observed among those patients with persistent severe neutropenia. Although AVA appeared to be superior to AV in these latter patients, the difference was accounted for largely by the maldistribution of episodes of pneumonia. There were six episodes of pneumonia in patients with persistent neutropenia who received AV but no episodes in patients who receive AVA.

All three regimens were well tolerated. The major toxicity was drug rash which occurred during 18 episodes (5 percent), primarily among patients who received AV or AVA (16 episodes). No patient developed nephrotoxicity due to these antibiotics. Eight patients developed superinfections, 4 caused by fungi, 1 by virus, and 3 by unidentified pathogens.

DISCUSSION

Aztreonam is an interesting new β-lactam antibiotic with broad-spectrum activity limited to gram-negative bacilli. A major advantage of this antibiotic is that its activity against gram-negative bacilli is comparable to that of an aminoglycoside, but it avoids the nephrotoxic potential of the aminoglycosides. In recent years, there has been a resurgence of infection caused by gram-positive organisms in cancer patients, hence, aztreonam must be combined with another antibiotic to provide sufficient coverage for initial empiric therapy in neutropenic patients. We selected vancomycin because it provides coverage against JK diphtheroids, *Bacillus* sp., *S. epidermidis*, and enterococci which are not uncommon pathogens in this patient population and usually are resistant to many other antibiotics. Furthermore, vancomycin has no activity against gram-negative bacilli, allowing an accurate evaluation of aztreonam against these infections.

Moxalactam plus ticarcillin was selected as a control arm in this study because it produced favorable results in a previous study.[8] In the current study, this regimen was effective against gram-negative bacillary infections, but only a few patients with these infections were assigned to it. Its major deficiency was against gram-positive infections in which the response rate was only 43 percent. None of the *S. aureus* infections responded to this regimen, which was not surprising, since neither of these antibiotics are effective antistaphylococcal agents.

The accepted practice has been to administer a β-lactam plus an aminoglycoside as initial empiric therapy for presumed infection in neutropenic patients. This practice has been based upon observations that synergistic combinations are more effective than nonsynergistic combinations.[9,10] Our previous studies have indicated that aminoglycosides are suboptimal in neutropenic patients[11,12] and in three separate prospective randomized trials, aminoglycoside-containing regimens have not been superior to double β-lactam regimens.[8,13,14] In the current study, the best results were obtained when amikacin was added to the AVA regimen. However, among documented infections, this difference largely was accounted for by a maldistribution of episodes of pneumonia and not to a higher efficacy of the AVA regimen. Although the numbers in each group were small, AVA conveyed no advantage over AV against gram-positive or polymicrobial infections. The difference among gram-negative bacillary infections was due to the failure of AV against two *Pseudomonas* infections. Other investigators have suggested that antibiotic combinations especially are necessary to treat *Pseudomonas* infections.[15] However, in a recent review of 410 episodes of *Pseudomonas* bacteremia, we found no difference in response rates between an antipseudomonal β-lactam alone and the combination of an antipseudomonal β-lactam plus an aminoglycoside.[16] Additional experience is needed to determine whether aztreonam alone is sufficient therapy for gram-negative bacillary infections, and especially *Pseudomonas* infections in this patient population.

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AZTREONAM: AN APPRAISAL

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Extensive clinical studies have shown that aztreonam, the first monobactam, is effective for urinary, respiratory, intra-abdominal, gynecological, and bone and joint infections. Aztreonam appears to be an effective agent that can be used singly or as combined directed therapy.

Aztreonam is the first clinically useful compound belonging to the class of beta-lactam antibiotics referred to as monobactams. It is logical to inquire why we need another class of antibiotics considering the proliferation of penicillins, cephalosporins, and aminoglycosides which have become available in the past decade. This article is an appraisal of this new agent in an attempt to evaluate its antibacterial, pharmacological, and clinical properties.

Major progress in the care of the critically ill patient has occurred in the past several decades. The importance of gram-negative infections was well recognized by the mid-1960s, but what is most important is that gram-negative infections have increased as a major nosocomial problem in spite of improvements in the prevention of nosocomial infections.[1] Indeed, analysis of data from the Centers for Disease Control reveals that *Escherichia coli*, *Klebsiella pneumoniae*, and *Pseudomonas aeruginosa* consistently are found to be the major pathogens involved in nosocomial infection.[2] Furthermore, species such as *Serratia marcescens*, *Enterobacter* species, *Providencia*, and *Citrobacter* account for an appreciable number of hospital-acquired urinary, pulmonary, and skin-structure infections.[2]

Aminoglycoside antibiotics have been used as initial therapy in a major number of suspected or proved serious gram-negative infections, particularly when *Pseudomonas aeruginosa* is a likely pathogen.[3] While the aminoglycosides are effective, they also are a cause of significant nephrotoxicity and ototoxicity. Furthermore, the aminoglycosides, while superb in the test tube, are less effective under a number of the clinical situations in which they are most needed. The small difference between efficacy and toxicity makes their use a problem for the physician who has to use the drugs only occasionally. Finally, the problems attendant on any hospital service, such as prompt collection and delivery of specimens, make the excellent serum assays of these drugs less useful in the everyday management of the critically ill patient. These factors have made the medicinal chemist and microbiologist search for two types of agents. The one type is a compound with broad spectrum activity and the other type is more directed at a particular group of organisms.

Aztreonam is a β-lactam, but its chemical structure differs from that of both the penicillins and cephalosporins by lacking either a thiazolidine or a dihydrothiazine ring, respectively.[4] An acidic sulfonic group is attached to the nitrogen of the β-lactam ring. There is an aminothiazolyl side chain and a carboxypropylimino group. The structural properties which correlate with the activity of the agent are a

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TABLE

Comparative In Vitro Activity of Aztreonam and New Cephalosporin Agents Against Major Pathogens

Organism	**Cefotaxime MIC$_{90}$**	**Ceftizoxime MIC$_{90}$ μg/ml**	**Cefoperazone MIC$_{90}$**	**Moxalactam MIC$_{90}$**	**Cefazolin MIC$_{90}$ μg/ml**	**Aztreonam MIC$_{90}$**
Streptococcus pyogenes (group A)	0.12	0.03	0.12	4	0.05	>32
Streptococcus agalactiae (group B)	0.1	0.1	0.25	4	0.1	>32
Streptococcus faecalis	>128	>128	>128	>128	>128	>32
Streptococcus pneumoniae	0.12	0.12	0.25	2	0.1	>32
Streptococci (alpha, non-hemolytic, not group D)	0.25	0.25	1	32	0.1	>32
Staphylococcus aureus	2	2	4	16	1	>32
Staphylococcus epidermidis	8	8	8	32	4	>32
Haemophilus influenzae	0.03	0.03	0.12	0.06	32	0.1
Neisseria gonorrhoeae	0.015	0.015	0.06	0.06	8	0.1
Neisseria meningitidis	<0.01	<0.01	0.05	0.05	0.01	0.1
Listeria monocytogenes	>128	>128	>128	>128	>128	>32
Escherichia coli	0.25	0.25	16	0.25	>128	0.2
Klebsiella pneumoniae	0.25	0.25	16	0.25	>128	0.2
Enterobacter aerogenes	0.2	0.2	2	0.2	>128	4
Enterobacter cloacae	8	8	32	4	>128	4
Citrobacter freundii	8	8	16	8	>128	4
Citrobacter diversus	0.5	0.25	1	0.5	8	0.1
Serratia marcescens	4	2	16	4	>128	4
Proteus mirabilis	0.1	0.1	1	0.2	16	0.1
Morganella morganii	2	0.5	4	0.5	>128	0.1
Providencia rettgeri	2	2	16	2	>12	0.1
Providencia stuartii	2	2	16	2	>12	0.1
Salmonella species	0.25	0.25	1	0.25	32	0.1
Shigella species	0.25	0.25	1	0.25	8	0.1
Pseudomonas aeruginosa	64	64	32	32	>128	16
Pseudomonas maltophilia	64	64	32	128	>128	>32
Pseudomonas cepacia	64	64	32	128	>128	>32
Pseudomonas fluorescens	64	64	32	128	>128	>32
Pseudomonas putida	>128	>128	>128	128	>128	>32
Acinetobacter	>64	>128	>128	>128	>128	>32
Bacteroides fragilis	64	32	64	16	>128	>32

methyl group at position 4 of the β-lactam ring to provide β-lactamase stability against plasmid β-lactamases and the carboxypropylimino group to yield anti-*Pseudomonas* activity while the aminothiazolyl structure provides activity against the *Enterobacteriaceae.* The lack of an adjoining ring structure has resulted in loss of activity against gram-positive and anaerobic species.

Aztreonam is similar to other β-lactams as it interferes with cell wall biosynthesis. Aztreonam binds preferentially to a β-lactam binding protein which is designated as penicillin-binding protein PBP3 in *Enterobacteriaceae.* Binding of an antibiotic to this protein results in production of long filamentous structures which ultimately lyse and die. Aztreonam does not bind to the PBPs of species such as staphylococci or streptococci, as do penicillins and cephalosporins, which explains its lack of activity against these species.

Aztreonam will inhibit the majority of *Enterobacteriaceae* at concentrations below 1 μg/ml.[5-7] Indeed, 90 percent of *Escherichia coli, Klebsiella pneu-*

moniae, Proteus mirabilis, Morganella morganii, Providencia rettgeri, and *Providenicia stuartii* are inhibited by 0.25 μg/ml. This in vitro activity is superior to agents such as ampicillin, carbenicillin, cefazolin, cefamandole, cefuroxime, and cefoxitin. Aztreonam has activity comparable to the third-generation cephalosporins, cefotaxime, ceftizoxime, ceftriaxone, cefmenoxime, ceftazidime, and moxalactam. It also inhibits *E. coli* and *Klebsiella* resistant to cefoperazone.[8]

Some isolates of *Klebsiella oxytoca* have aztreonam MICs of 8 μg/ml and certain *Citrobacter freundii* and *Enterobacter aerogenes* and *Enterobacter cloacae* are resistant, MIC > 16 μg/ml, although 75 percent of the isolates of these species would be inhibited by < 4 μg/ml. Aztreonam inhibits 50 percent of *Pseudomonas* aeruginosa at concentrations of 4 μg/ml and 90 percent at 16 μg/ml.[5] It is superior in activity against *Pseudomonas* to cefoperazone, moxalactam, and cefsulodin and generally equal or one dilution less active than ceftazidime. Since it is much more β-lactamase stable than ticarcillin, piperacillin, or cefoperazone, there is less of an inoculum effect than is seen when these agents are tested against *Pseudomonas*. Aztreonam does not inhibit the majority of *Acinetobacter* and *Pseudomonas maltophilia*, and 50 percent of other *Pseudomonas* species also are resistant.

β-LACTAMASE STABILITY

Aztreonam is not hydrolyzed by the major plasmid and chromosomal β-lactamases.[5,6] There is minor hydrolysis of aztreonam by the β-lactamase called K-1 present in some *Klebsiella oxytoca* isolates and by the *Pseudomonas cepacia* β-lactamases. Like many of the cephalosporins which are β-lactamase stable, aztreonam binds to enzymes of the Richmond-Sykes type I class and is to a degree an inhibitor of these enzymes; albeit less efficient than are the β-lactamase stable penicillins. Aztreonam does not function as an efficient inducer of β-lactamases, although it can induce β-lactamases in some *Enterobacter cloacae*.

PHARMACOLOGICAL PROPERTIES

Aztreonam can be measured in serum, urine, and other biological fluids by both a microbiological assay and high-pressure liquid chromatography. These methods have shown that there are no major metabolites. Aztreonam administered orally is not absorbed. It is well absorbed by the intramuscular route with peak serum levels after 500 mg of 22 μg/ml at 1 hour and levels of 1.7 μg/ml still detected at 8 hours.[9] Serum levels after 1 g are peak of 46 μg/ml at 1 hour and 3.5 μg/ml at 8 hours with 0.65 μg at 12 hours. We have shown that 1 g administered by 30 min infusion will produce serum levels at the end of infusion of 165 μg/ml and at 2 g, a level of 255 μg/ml.[10] Levels at 8 hours are 3.5 and 8.5 μg/ml after 1 and 2 g, respectively. Bolus infusion of 1 g will produce a 5 min level of 125 μg/ml and 242 μg/ml after 2 g. Multiple doses in normal subjects and patients with normal renal function have not shown accumulation of aztreonam. The half-life in individuals with normal renal function ranges from 1.5 to 2 hours. Aztreonam is 56 percent bound to serum protein and produces a steady state volume of distribution of 0.18 L/kg. Serum protein binding is decreased moderately with depressed renal function as is seen with other β-lactam drugs.

The drug is cleared primarily by glomerular filtration, hence serum half-life increases as creatinine clearance falls with a $T_{1/2}$ of six hours in individuals who have creatinine clearances less than 10 ml/min.

Urine concentrations, after 500 mg administered intramuscularly, range from 200 to 700 mg in the first 2 hours and are concentrations of 20 to 40 μg/ml 8 to 12 hours after the injection. After 2,000 mg administered intravenously levels of 5,000 to 7,000 μg/ml are attained with levels of 10 μg/ml still present at 24 hours.

Levels of aztreonam in other body fluids indicate that pleural fluid and synovial fluid exceed the MICs of most *Enterobacteriaceae* for at least eight hours after a 1 g intramuscular or intravenous dose. Cerebrospinal fluid levels of 2 to 20 μg/ml have been recorded in individuals with purulent meningitis and bone levels have been above the MICs of most *Enterobacteriaceae* and many *Pseudomonas aeruginosa*.

CLINICAL USE

Aztreonam has been evaluated in both open studies and in controlled comparative studies in the United States, Europe, and Japan. The programs from these different parts of the world have shown similar clinical and microbiological responses. Aztreonam has cured bacteremia due to *E. coli, K. pneumoniae, P. mirabilis, S. marcescens, P. aeruginosa, Enterobacter, Citrobacter, Proteus,* and *Providencia* species. Of the 101 patients with bacteremia treated in the United States, 94 percent were cured. Sources of the bacteremia have been the urinary tract, pulmonary system, skin infections, gynecological infections, and intra-abdominal infection. Comparative studies in non-neutropenic patients have shown that aztreonam clinically and bacteriologically is comparable to aminoglycosides such as gentamicin, tobramycin, and amikacin. Clinical and microbiological results also are comparable to those previously reported for third-generation cephalosporins such as cefotaxime, ceftizoxime, and moxalactam.[11-13]

Aztreonam has been employed successfully as therapy of gram-negative pneumonia used either singly or in combination with clindamycin. It has proved equal or superior to a tobramycin-clindamycin treatment program. We particularly have been impressed with the response of patients with *Pseudomonas* respiratory infections which have failed to respond to other programs which combined an anti-*Pseudomonas* penicillin and an aminoglycoside.[14]

Use of aztreonam by our group to treat pulmonary exacerbations of cystic fibrosis has been favorable with 18 of 21 patients treated showing a clinical response. In such patients, the organisms persist in the sputum as they do with other forms of therapy and the concentrations needed to inhibit the bacteria can increase to resistant levels. Studies of combination of aztreonam and tobramycin to treat cystic fibrosis patients are in progress.

The response of patients with intra-abdominal infections treated with clindamycin and aztreonam has been comparable to treatment with clindamycin and an aminoglycoside. This also has been true for gynecological infections.

There have been many studies on the use of aztreonam as therapy of urinary tract infection. These have ranged from single dose intramuscular injection to multiple dose therapy for complicated infection. Microbiological cure rates have been good with response to aztreonam equivalent or superior to the response seen for the comparative agent. Twice-daily dosing programs have proved adequate.

Although the overall number of patients with septic arthritis or osteomyelitis treated with aztreonam is not large, the preliminary reports indicate that aztreonam is effective against both *Enterobacteriaceae* and *P. aeruginosa* including isolates resistant to β-lactam antibiotics and aminoglycosides.

UNTOWARD EFFECTS

Aztreonam has been shown not to cross-react with IgE anti-penicillin antibodies.[15] The number of patients with proved immediate hypersensitivity to penicillin who have received aztreonam has been small so one would use the agent with caution in such patients. No serious hematological or renal effects have been noted. Minor increases in serum transaminase levels have been reported, but these have returned to normal. Superinfection with *Streptococcus faecalis* has been noted in a few patients. No abnormalities of clotting such as those seen with cefamandole, cefoperazone, or moxalactam have been reported.[16] Also, the combination of aztreonam with nafcillin, oxacillin, ampicillin, vancomycin, clindamycin, or metronidazole has not resulted in any increase in adverse effects.

OVERALL USE OF AZTREONAM

There are several areas in which use of aztreonam seems logical. Organisms known to be resistant to multiple antibiotics can be treated directly with aztreonam as single directed therapy, avoiding more toxic agents.

In mixed gram-positive and gram-negative bacterial infections, aztreonam can replace the aminoglycoside component. In settings of mixed bacteriology when there is suspected drug-resistant *Enterobacteriaceae* and/or *Pseudomonas aeruginosa*, aztreonam can be combined with an antigram-positive agent or an anti-anaerobic agent.

Aztreonam has proved to be effective, safe therapy for serious and life-threatening infection due to multiply-resistant aerobic gram-negative bacteria. It should be used in combination to treat infection if the etiology is not known, particularly in the immunocompromised, neutropenic patient. Doses of 1 g every 8 to 12 hours will treat infections due to the majority of *Enterobacteriaceae*, but doses of 2 g 8 hourly would be preferred for systemic *Pseudomonas* infections. Urinary infections due to gram-negative bacteria can be treated with an intramuscular dose of 500 mg twice or once daily. Dosage should be adjusted in renal failure. Clearly, aztreonam is a useful addition to the physician's armamentarium of antimicrobial agents.

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AZTREONAM: A ROUNDTABLE DISCUSSION

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A roundtable discussion concerning the beginning of a whole new family of monobactams was held in 1984. The participants covered a varied program on the new antibiotic, aztreonam.

Dr. Smith: We are enthusiastic about a new compound discovered in New Jersey. Dr. Richard Sykes of the Squibb Pharmaceutical Corporation has developed a new family of compounds.

Dr. Sykes: In 1978, we made a commitment to look for novel types of β-lactam-containing molecules in nature. We devised a completely new technology based on the fact that β-lactams have one thing in common; they induce β-lactamases in certain gram-positive and gram-negative bacteria. Utilizing this technology, we examined soil and water samples from all over the world. The first positive samples to come into the laboratory were taken in the Pine Barrens of New Jersey. These samples contained organisms identified as *Chromobacterium violaceum*. The *Chromobacterium* strains produced a single cell nucleus β-lactam molecule that previously had not been identified in nature. This molecule led us to a synthetic derivative of the nucleus 3-aminomonobactamic acid which subsequently was modified to produce a whole series of monobactams. These molecules were called monobactams because chemically they were monocyclic rings produced by bacteria and the monocyclic rings were β-lactam in nature. From that chemical series came aztreonam, so called because the molecule is produced synthetically from threonine and the four-membered β-lactam ring is referred to as an ajetidine ring. We have manipulated the monobactam nucleus to produce other types of molecules with different properties than aztreonam. We also are looking at compounds that would have the potential for treating gram-positive and anaerobic infections, and to produce molecules with very high activity against organisms such as *Acinetobacter*, *Enterobacteriaceae*, and *Pseudomonas*.

Dr. Smith: We need to emphasize that this is entirely New Jersey's product: the soil from the Pine Barrens and the research from Princeton. Not since streptomycin, with Waxman and Rutgers, have we had such an interesting finding in New Jersey.

Dr. Neu: Aztreonam is a very interesting compound because of its excellent activity against gram-negative bacteria. It does not have activity against gram-positive bacteria nor against anaerobic species. One should realize that gram-negative micro-organisms, i.e. *Escherichia coli*, *Klebsiella pneumoniae*, *Proteus mirabilis*, and organisms such as *Pseudomonas aeruginosa*, have become important causes of hospital infections. For such infections we often have utilized combination therapy that is a β-lactam antibiotic, i.e. penicillin or cephalosporin and aminoglycoside. Aztreonam has provided us with directed therapy in the β-lactam class of antibiotics, and the ability to have a safe drug with minimal toxicity—a marked difference between the activity and toxicity in contrast to the aminoglycoside compounds. There are many areas in which a compound such as aztreonam would prove useful. As we know, urinary tract infections, the most common nosocomial infection in the hospital, usually are due to gram-negative organisms. A drug like aztreonam would inhibit virtually 95 percent of these organisms—possibly 99 percent—at concentrations less than a microgram per milliliter. You would have one drug rather than combination therapy. Most importantly, you would have a compound that would not alter the microbiological flora that protects us against fungal or other opportunistic infections that might develop in the hospital. We have used aztreonam for patients with respiratory infections and it has proved to be very useful. In situations such as the skin structure infections, a drug like aztreonam would be directed against specific organisms. Aztreonam is a useful compound with a specific activity in an era in which we are trying to streamline and narrow our choices of antibiotics for infectious diseases.

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Dr. Bendush: In 1981, after aztreonam had been characterized in the laboratory, we studied its pharmacokinetics. We were intrigued by the fact that the compound had β-lactamase stability, but we wanted to get more information on tissue distribution, metabolism, and excretion. All of these characteristics led us to conclude that the dosage of 0.5 g to 2 g up to four times a day should cover the range of the organisms that could be expected to produce infectious diseases in the target population. With this information, we put together a clinical trial program in 1981 with Drs. Neu, Hewitt, and Weinstein. We decided to study two types of patients: those in whom single-dose therapy would benefit, and those in whom multiple-dose therapy would benefit. In the initial program, we studied patients who had uncomplicated cystitis or gonorrhea, using single 1 g doses. Those studies proved, as we predicted, that the drug was effective. In fact, about 10 percent of the patients with uncomplicated gonorrhea had penicillinace-producing organisms. The overall microbiologic cure rate in that group of patients with gonorrhea had been in the range of 97 percent. The other group of patients we studied were those with uncomplicated cystitis, and 0.5 g given as a single intramuscular injection proved to be effective with a high cure rate around 83 percent, which was comparable with an amoxacillin control group. We also were interested in how the drug would perform in patients with serious illnesses and in patients with *Pseudomonas* infections. The program was expanded both to control and open studies. Today we have about 9,000 patients worldwide that have been treated in all of the clinical trial programs. In patients with serious infections, we started combination therapy or with a therapeutic regimen that would be supplemented by a second drug, such as clindamycin, to cover the anaerobes and organisms in the gram-positive end of the spectrum; once we were able to determine what the patient had, then the second drug could be discontinued. The concept of targeting or directing therapy, which is commonly employed today, was one that we began to study in these clinical trial programs. We have been able to demonstrate an overall microbiological cure rate of 78 percent in patients with lower respiratory tract infections; for urinary tract infections, septicemia, and intra-abdominal infections, cure rates have ranged as high as 99 percent. Aztreonam has proved to be a very valuable antibiotic used either alone or in combination, and the predictions for the drug in the microbiology laboratory have been borne out by the clinical results we have achieved.

Dr. Smith: This drug has a very narrow spectrum that does not kill anaerobes and it does not cross react with other β-lactamases. Dr. Neu, would it be fair to say it is safe to use in individuals allergic to penicillin?

Dr. Neu: I think the problem of allergy to penicillin is one that needs re-evaluation at the present time. One, though, would not want to recommend that someone who had acute anaphylaxis to penicillin be treated with a monobactam compound.

Dr. Smith: Has there been any case reported with pseudomembranous colitis with aztreonam?

Dr. Bendush: We have two patients in whom diarrhea developed during therapy and the clinical diagnoses were pseudomembranous colitis; yet this was thought to be the result of previous antibiotic therapy. We noted that about 2 percent of patients in the clinical trial data base had various gastrointestinal side effects: nausea, vomiting, and diarrhea.

Dr. Smith: Is it safe to use this compound in central nervous system diseases with the blood brain barrier?

Dr. Bendush: In the clinical pharmacology and ongoing studies, we have treated 11 patients with meningitis. We had 2 failures because of infected shunts.

Dr. Smith: We are interested in osteomyelitis and believe aztreonam has unique properties for treating chronic osteomyelitis due to *Pseudomonas*, another gram-negative organism. It is safe to use for a long time and the overgrowth for bowel flora and other flora is small—so aztreonam is an ideal treatment for *Pseudomonas* osteomyelitis.

Dr. Bendush: Concerning osteomyelitis, we are looking at the tissue and fluid distribution for aztreonam. We are gratified to see that the good levels in bone and joint fluid in infected joints were in the range of 80 μg/g and bone levels were in the range of 20 μg/g. In the trials, we have treated 19 patients and 18 have responded well.

Dr. Smith: Would you use aztreonam in the treatment of serious *Pseudomonas* infections?

Dr. Moellering: I am concerned about the possibility of emerging resistance. If I were using aztreonam for a life-threatening *Pseudomonas* infection, my inclination would be to use it in combination. It is my hope that we may be able to define some situations in which we can use this drug alone for serious *Pseudomonas* infections.

Dr. Smith: What is your estimate of where aztreonam will be in the next five years with respect to the new β-lactamase antibiotics?

Dr. Moellering: There is a multitude of new β-lactam antibiotics on the market. This makes it difficult for the clinician to keep up with these drugs—much less sort them out. One over-riding characteristic of all these drugs is that they are broad-spectrum agents. Many clinicians feel more comfortable with a broad-spectrum drug, but there are problems with this, including the destruction of normal host flora. Because aztreonam is a narrow-spectrum agent, with little activity against anaerobes and gram-positive organisms, it has a less deleterious impact on the normal flora. This potential advantage makes aztreonam stand out among the other new agents. In addition, aztreonam is unique among β-lactams because of its chemical structure—it does not create cross sensitivity and does not cross react in patients who are proved allergic to the classic penicillins and cephalosporins. Thus, we have an agent which can be utilized to treat serious gram-negative infections in patients who are allergic to β-lactams.

Dr. Smith: Knowing that aztreonam is a β-lactam, would you expect to see any bleeding problems or antabuse-like effects?

Dr. Bendush: The bleeding problem and the antabuse-like effects of the modern β-lactams have been seen in those that contain methyltetrazole—such as moxalactam. Aztreonam has no such substituted position on the ring, so you would not expect to see these side effects.

Dr. Neu: One of the merits of aztreonam is that it can replace aminoglycoside use. For most hospitalized patients, you need an antigram-negative agent for pneumonia after abdominal or thoracic surgery, for urinary tract infection following gynecological surgery, or for infection following placement of a catheter. In many of these situations, a physician utilizes combination therapy of an aminoglycoside. I wonder whether the physician, who is not conversant with these drugs, would be safer with aztreonam. Aztreonam is easier to use than an aminoglycoside if the physician does not understand blood levels. Daily creatinine and BUN levels are not needed and its use will be cost effective.

Dr. Smith: We all recognize that there are tremendous changes taking place in the hospital environment and that there is going to be more home use of antibiotics. Aztreonam offers a margin of safety in this area. We have had no reports of ototoxicity. We have monitored renal function and there have been no problems with renal impairment or changes in renal function parameters. We feel very comfortable that aztreonam, like other β-lactams, is going to be safe from the standpoint of renal and ototoxic effects. We have continued to watch these patients with respect to bleeding. What is your experience with allergic reactions?

Dr. Bendush: In Italy, a physician was skin testing a patient who received over 100 mg of aztreonam intracutaneously and developed an anaphylactic reaction. He was treated and responded promptly. There is no additional followup and no past history of drug allergy.

Dr. Moellering: Do you have any data on neutropenia with this drug?

Dr. Smith: We have looked at changes in all the blood elements—increases and decreases—and we have compared those changes across the board with the control drugs. If one defines neutropenia or leukocytosis using standard terminology, we have referred to that as marked deviations. There have been patients who have showed both increases and decreases in all the blood elements. But there have not been patients who developed neutropenia. Also, one can get a good estimate of how a compound will do by the attitude of the house staff. At our institution, the house staff loved the compound.

Dr. Moellering: There has been a lot of enthusiasm for aztreonam because it can be used in a variety of therapeutic programs that are tailored to meet the clinical situation. One of the most attractive features of this drug is its flexibility. Aztreonam has a narrow spectrum and it fits in nicely with certain other agents, i.e. clindamycin, vancomycin. Aztreonam gives us the opportunity to do something that we preach and seldom practice: to actually narrow the spectrum of a given initial treatment regimen once the affecting organism is known and to truly use a rifle instead of a shotgun. With aztreonam we can do that if it is used properly.

Dr. Smith: Please tell us where it binds in its protein binding site in comparison to the other cephalosporins and β-lactam drugs?

Dr. Sykes: Penicillins tend to bind to PBP1 complex and then to PBP3. Cephalosporins bind preferentially to PBP3 and then to the PBP1. Aztreonam binds almost exclusively to PBP3. Aztreonam is rather unique in that respect—it binds at very low concentrations, less than 0.1 μg with 100 percent binding affinity for PBP3, which results in an elongation of the cell.

Dr. Smith: I think we have covered a great deal about this exciting new compound whose origin was in New Jersey and whose development is by a New Jersey corporation. Aztreonam seems to have greater safety than other compounds, a lower incidence of pseudomembranous colitis, and a decreased cross allergenicity. In addition, aztreonam will have a special role in home health nursing care and in the hospital setting particularly for nosocomial infections. Aztreonam has very interesting pharmacokinetics. It has a very interesting ability to bind to protein sites. The most interesting part of aztreonam is the beginning—a beginning of a whole new family of monobactams.

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